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ON

BRONCHIAL ASTHMA

ITS PATHOLOGY AND TREATMENT

BY

J. B. BERKART, M.D

Late Physician to the City of London Hospital for Diseases of the Chest

SECOND EDITION

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PREFACE

TO THE SECOND EDITION.

THE present edition is entirely re-written, and differs from the former one, in that it mainly contains the records of observations, which have been made during a period of ten years. All discussions of, what may be called, contentious matter have been, as far as possible, avoided in the hope, that the facts themselves will sufficiently show, whether the current doctrines of pathology be tenable or not. So, also, subjects that are well known, or that may elsewhere be found, have been omitted. Throughout the work, I have constantly endeavoured to be as concise as is consistent with the nature of the task.

The opinions which I ventured to adopt years ago have been fully confirmed by a more extensive clinical experience. They are modified only in so far as the term Asthma is here used in a more restricted sense, and applied to a definite form of disease.

No one knows better, than I do myself, the many imperfections of the present treatise. For some of them I myself am to blame; for others, the peculiar circumstances, in which asthmatics are generally seen, are chiefly responsible.

I must not omit to express my gratitude to Dr. Hueppe, of Wiesbaden, for his great kindness in sacrificing his well-earned holidays in order to help me in my bacteriological enquiries. The time at his disposal, and at mine, was, however, too short to carry them to a satisfactory conclusion.

WIMPOLE STREET, W.,
May, 1889.

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CHAPTER I.

REVIEW OF THE PATHOLOGY OF BRONCHIAL ASTHMA.

Period before the Introduction of Physical Diagnosis—Corvisart—Laënnec—Andral—Physiological Inquiries into the Bronchial Contractility—Asthma a Symptom of Pathological Changes—Theories of:—Spasm and Paralysis of the Bronchi; Bronchial Spasm only; Spasm from Humoral Causes; Spasm from Bronchial Exudation; Spasm of the Diaphragm; Vaso-motor Disturbances.

Period before the Introduction of Physical Diagnosis.

The history of Bronchial Asthma is said to be traceable to the remotest periods of antiquity. Certain passages in the the Bible and in the Iliad have been adduced as proofs, that the disease was, then, already known.[1] Hippocrates, Aretæus and Celsus are alleged to have given graphic accounts of its clinical aspect; and Willis, Van Helmont and others, to have correctly interpreted its nature.[2]

It may, at once, be conceded, that Asthma is, in all probability, as old as the human race itself. If so, there can be little doubt that it, at all times, attracted the attention of observers. Still, there is no evidence, that the references to it, which may be found in the works of the ancient, and of even the more modern writers, are, in any way, applicable to the Asthma of to-day. For, up to a comparatively recent date, it was customary to distinguish and to name diseases according to the one or the other of their striking clinical features. The term "Asthma" was, thus, used in a generic sense—to denote all forms of dyspnœa, which fluctuated in their intensity; and therefore it comprised a promiscuous group of affections, which had nothing in common but the one conspicuous symptom.

---

[1] Bergson, *Das krampfhafte Asthma*, 1859, p. 6, *seq.*

[2] Théry, *De l'Asthme: Ouvrage couronné par l'Acad. Imp. de Médecine*, 1859.

CORVISART.

The form of dyspnœa, which is now called "nervous Asthma," is discernible only since the introduction of physical diagnosis. At the beginning of the present century, the current species and varieties of it were, still, very numerous. Some of them, however, were soon shown to be mere symptoms of definite organic lesions. The first step in that direction was taken by Corvisart.[1] He had, meanwhile, rescued from oblivion Auenbrugger's invention of percussion; and by the practice of it, he was enabled to recognise, during life, certain forms of diseases of the heart, which were, as yet, either imperfectly or not at all known. Rightly he inferred, therefore, that the dyspnœa in those cases was not due, as was generally supposed, to a disturbance of innervation, but that it was the necessary consequence of appreciable anatomical changes. Not unnaturally, he suspected, that other species of "nervous Asthma" might, similarly, betray themselves as symptoms; but his supposition, though well founded, could not be confirmed by percussion alone.

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[1] *Sur les Maladies et les Lesions Organiques du Cœur*. 1806.

LAËNNEC.

The invention of auscultation brought physical diagnosis to the highest degree of perfection. In the master-hands of Laënnec, the whole thoracic pathology at once assumed a totally different aspect. Most of the prevalent forms of the "asthme des praticiens" ceased, henceforth, to be regarded as idiopathic affections. Some of them were found to be symptoms of such well-defined diseases as "catarrh sec" and "emphysema," the pathology of which was, notwithstanding their frequent occurrence, till then almost unknown.

But in this sweeping measure, he could not comprise two forms of paroxysmal dyspnœa—Asthma with puerile respiration, and spasmodic Asthma.

Although he had observed that the Asthma with puerile respiration generally occurred in individuals, who were subject to "mucous catarrh,"[1] it was, nevertheless, doubtful to him, whether such catarrh satisfactorily accounted for the paroxysmal character of the dyspnœa. If he could convince himself, that it produced a transient obstruction of the bronchi, the problem would readily be solved. But he had no evidence on it of any kind. The sputum was not particularly tenacious, and expectoration was always easy. The possibility of complications on the part of the heart, he never considered. He felt constrained, therefore, to admit that those attacks of breathlessness, probably, arose from an unknown disturbance of innervation.

To decide on the nature of the spasmodic form was, however, a more difficult task. The stethoscope which had, all along, been his infallible guide, seemed to fail him in this instance. He had, indeed, seen, in most of the cases that had come under his notice, emphysema, pituitous, or dry catarrh. But he had also met with some, in which such affections could not be detected; and, even if these had been invariably present, they might, possibly, be answerable for the intensity, yet would not explain the fitful occurrence of the dyspnœa. It was evident to him, that permanent organic lesions could not be the immediate cause of it; but that the rapid onset and the sudden cessation of the attack presupposed one, that came and went in a similar manner.

---

[1] *Traité de l'Auscultation, etc.*, Paris, 1879, p. 519 *seq.*

Moreover, he had to consider also, that trivial circumstances—as, for instance, sleeping in a dark room, or in one with doors closed—had been alleged to have provoked the dyspnœa. It is but natural, that patients should seek to account for the origin of such formidable accidents in order to avoid their recurrence; but in their anxiety to arrive at a conclusion, they generally fasten on anything, accidentally precedent, which especially strikes their fancies. Laënnec, however, attached to their opinions an undue importance, forgetting that, in such matters, their judgment must be, more or less, warped.

Convinced as he was of the correctness of their assertions, he had, therefore, to interpret the bearings of the supposed causes upon the pathology of the disease. None of those above mentioned could produce either congestion or exudation; consequently, their influence could be exerted only in a manner, that led to no structural changes. Thus he was induced to infer, that spasmodic Asthma was not, necessarily, dependent on organic lesions of the lungs.

The results of a post-mortem examination in a fatal case of Asthma, which had occurred in Andral's practice, seemed fully to support the view, which he had adopted.[1]

"A man, æt. 40, had suffered, for a long time, from a large ulcer of the left leg. The tissues of the foot were greatly swollen and of almost stony hardness. The limb, with its dirty greyish colour, presented, in many respects, the appearance of elephantiasis. Usually, there was abundant discharge from the ulcer. For the last five or six years also, the patient had been subject to a slight cough; but he had never had dyspnœa nor pain in the chest.

"By mistake, he was admitted into the medical ward and remained there for several days; but when about to be transferred to the surgical side of the hospital, he was suddenly seized with great dyspnœa. It was noticed, on the occasion, that the discharge from the ulcer was much less than it had been before. The patient was sitting in an arm-chair, gasping for breath; and, in a state of inexpressible anxiety, he begged to be relieved of the enormous weight, which compressed his chest, threatening suffocation. Inspiration was short, very frequent, and at times convulsive. The pulse was rapid and thready. The chest was frequently examined, but always with negative results. No cause of this formidable accident could be detected. The thorax was resonant everywhere, except on the left and behind, over a space of a few inches, where a slight dulness was noticed. At that place, respiration was feeble and accompanied by rhonchi, while over the rest of the lungs, it was quite normal. The heart and the large vessels also seemed to be healthy.

"Thus no clue whatever was obtained as to the nature of the dyspnœa, which constantly became more intense. Bleeding, blistering, etc., were of no avail. The next morning, the dyspnœa had so much increased, that the patient was in imminent danger of asphyxia. The question was then raised, whether it might not proceed from laryngeal obstruction, as the symptoms somewhat resembled those of œdema of the glottis. Tracheotomy was consequently performed on the chance of preserving the patient's life; and with Professor Roux as operator, it is needless to add, that all due precautions were taken to insure the free entrance and exit of air to and from the tracheal opening. No amelioration, however, followed the operation. The dyspnœa persisted, and the patient died the same evening.

---

[1] *Op. cit.*, p. 525.

"On post-mortem examination, the lungs were healthy and crepitating, except the lower lobe of the left lung, which was in a state of hepatisation. In the bronchial mucous membrane, there were small red patches. The heart and large vessels were normal. No pathological changes could be found in the head and the abdomen. The subcutaneous tissue of the leg had undergone lardaceous thickening, and the tibia was necrosed."[1]

It is impossible to form, from the report of this case, an accurate idea of its nature. This much, however, is certain, that it was not one of what is generally understood by "spasmodic Asthma." But Laënnec thought otherwise. He was satisfied with the anatomical and the clinical diagnosis, which seemingly confirmed his previous supposition. Therefore, the chief and primary cause of Asthma could only be a "sudden alteration of the nervous influence."

The bent of his mind was, however, by no means towards the framing of hasty theories. Although he believed the facts, upon which he argued, to be fully warranted, he was yet not free from doubts on the correctness of his inference. But, while he readily granted to the nervous system the principal share in the production of the dyspnœa, he expressly and significantly added, that, as a rule, several causes contributed towards it.[1]

---

[1] Andral, *Clinique Médicale*, t. i., p. 250, 1829.

Nothing now remained for him but to explain the manner, in which the supposed alteration of the nervous system manifested itself. From the researches of Reisseisen it was known, that the bronchi were provided with muscular fibres. Their function, however, had not yet been ascertained. Laënnec, nevertheless, reasoned, that because they were muscles, they were, necessarily, contractile, and therefore liable also to spasm. Hence, he concluded that, most likely, the immediate cause of Asthma was a spasm of the muscles of the bronchi and of those of the alveoli, in which he gratuitously assumed their presence.

Thus came Asthma to be admitted into the nosology of to-day as a substantive affection of nervous origin. In point of fact, however, that theory had been held for centuries, and had proved its usefulness in many ways. For a time it seemed, as if the rise of pathological anatomy threatened its existence. But the sanction of Laënnec's authority restored its waning influence and secured it in its place. And this place it has maintained ever since, even against the most convincing arguments.

It is important, however, to note that the chapter, which Laënnec devoted to the consideration of "spasmodic Asthma," chiefly consists of his theoretical reflections on the

[1] *Op. cit.*, p. 528.

[2] *Op. cit.*, p. 521.

origin of the dyspnœa. It contains no records of clinical observations; no account of subjective and objective symptoms during the paroxysm; no mention, that he had ever had, in the latter, the opportunity of using the "cylindre." Rather it appears that, like most practitioners, he had seen asthmatics, generally, in the intervals between their attacks; so that, what he knew of the physical signs, he had learnt only then, and what he reported of the causes of the disease, had been "communicated" to him. But free intervals and asthmatic paroxysms differ so totally from each other, that any deduction from the former as to the latter must, necessarily, be imperfect.

ANDRAL.

No one, perhaps, had better reasons, than Andral, to suspect the alleged independence of Asthma on organic changes, and consequently the current theory of its nature. The following case, which had occurred in his own practice, ought to have, convincingly, shown him that, even on post-mortem examination, the real cause of a dyspnœal paroxysm may, readily, be overlooked; and that its nature may thus become the subject of idle speculation.

"A dock labourer, æt. 53, was admitted into the Charité Hospital on account of acute rheumatism. He had also suffered for about two months from severe cough and from difficult expectoration of a very tenacious sputum.

"On examining the chest, there was good resonance everywhere; vesicular breathing was distinct over the whole left side, but over the upper and middle lobes of the right side, there were mucous rhonchi. There was, however, no dyspnœa.

"One day, during a severe fit of coughing, the patient was suddenly seized with a violent attack of dyspnœa, which lasted without intermission till the next morning. Asphyxia then seemed imminent. His face was swollen and cyanotic and his extremities were livid. No pulse could be felt at the wrists. He spoke with difficulty only a few words, begging to be relieved of the enormous weight which he felt in the right mammary region and which threatened to suffocate him. The physical signs during the attack were the following: forcible expansion of the chest; good resonance everywhere; puerile respiration on the left; absence of vesicular breathing and of all adventitious sounds, from the right clavicle down to below the nipple and in the right supra-spinous fossa; mucous rhonchi towards the right base behind. The diagnosis, which at the time seemed the most probable, was emphysema. But scarcely was the examination finished, when the patient expired.

"On post-mortem examination, nothing abnormal was found in the cranial and the abdominal cavities. The heart was normal in size, and its right side contained black and coagulated blood. The lungs were congested, but buoyant when thrown into water. Larynx and trachea were normal."

To this report Andral adds :—

"We remained in complete ignorance as to the nature of the symptoms and as to the cause of the dyspnœa. We could not find anything to explain the absence of all respiratory sounds, which we had observed, during life, in the upper portion of the right lung, and had attributed to emphysema. But on opening the right main bronchus, the blunt point of the scissors encountered, at the origin of one of its larger branches, a mass of thick, almost solid mucus, by which that tube was completely occluded. The plug was gradually tapering, and was prolonged into the next division of the bronchus, the mucous membrane of which was of a deep red colour.

"We may be astonished to find," Andral proceeds, "that such serious symptoms resulted in this case from the obstruction of so small a portion of the lungs, while, in a large number of cases, disease of the organ may be so far advanced as to render it quite unfit for its function. Yet life not only is sustained for a long time, but there is, frequently, not even a great amount of dyspnœa. The reason is, that, in the one case, the respiratory surface is gradually diminished, whereas, in the other, the reduction was sudden.

"Let me also remark that, if in this case, the inspection of the lung had not been carefully made, one might have been led, in the absence of appreciable lesions, to regard it as one of 'nervous Asthma'; and the dyspnœa, which was, in reality, entirely due to a mechanical cause, would, most probably, have been considered as a remarkable instance of a metastasis of rheumatism to the lungs."[1]

This valuable lesson was, however, forgotten as soon as it was learnt. There were not the least grounds for supposing, that this case was at all isolated, as regards its pathology. But when he observed the one previously mentioned,[2] in which, it may be permitted to say, the post-mortem examination had *not* been made with all the requisite care, he concluded that, because no anatomical lesions were found, therefore, none existed. So firm was his conviction on this point, that he exclaimed, "If ever there was a case of purely nervous Asthma, surely it was this."

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[1] *Op. cit.*, tom. i., p. 215 *seq.*

[2] *Vide* p. 5.

He assumed, as the immediate cause of the dyspnœal paroxysm, a bronchial spasm, which, in its turn, was due to an abnormal irritability of the vagus and of the phrenic nerves. He based this theory upon the cases he had met with, in which orthopnœa had resulted from the pressure upon those nerves by enlarged bronchial glands, the lungs being, otherwise, intact.

PHYSIOLOGICAL INQUIRIES INTO THE BRONCHIAL CONTRACTILITY.

The opinions of Laënnec and of Andral were, upon the whole, fully endorsed. The more familiar practitioners became with the new method of clinical investigation, the more accurate became their diagnosis; and the more convinced were they, that there was one form of paroxysmal dyspnœa, which was not due to organic lesions, but which, on account of its clinical peculiarities, could be nothing else but a nervous affection. That theory, therefore, met with ready acceptance; the only drawback to it was the question of the bronchial spasm.

Hitherto, a spasm of the bronchial tubes had been, confidently, asserted as the immediate cause of the dyspnœa, while as yet no reliable evidence existed even of their contractility. The physiological data on the subject were scanty and inconclusive. All that was known was that, on chemical and mechanical irritations, they contracted in some, but failed to do so in other experiments.[1] Electricity, applied to the main bronchi and to their subdivisions, had no effect whatever, but produced, in those of intermediate size, a gradual narrowing to almost complete occlusion.

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[1] *Vide* the account given by Henle, *Ueber Tonus, Krampf u. Lähmung d. Bronchien, etc., Zeitschrift f. rat. Med.*, Bd. i., 1844.

At that stage, the matter was taken up by C. J. B. Williams;[1] and, henceforth, the pathology of the disease passed, to a great extent, from the bedside to the laboratory. In a series of carefully conducted experiments, he conclusively showed, that the bronchi contracted on mechanical, chemical and electrical stimulations. To obtain this result, however, it was necessary, that the lungs should not be inflated; for the contractility was not observed when they were distended; and, even after the expulsion of a certain volume of air, it was less pronounced than in a state of collapse.[2] But Williams did not succeed in demonstrating the innervation of the bronchi. Irritation of the vagus had no influence upon their calibres, and he, therefore, concluded that "the muscular fibres seem not to be excitable through the nerves of the lungs."[3]

Longet[4] confirmed in the main the conclusions, which Williams had arrived at, and solved, moreover, the moot point as to the innervation of the bronchi. He showed, that irritation of the vagus caused them to contract, whereas section of it led to emphysema of the lungs. He saw, by means of a magnifying glass, the narrowing of even the smallest bronchioles; but he could not satisfy himself of a similar occurrence in the air-vesicles. Still, he argued, because section of the pneumogastric nerve produced emphysema—*i.e.* distension of the air-vesicles—therefore, they must contain muscular fibres, which, if liable to paralysis, were liable also to spasm.

---

[1] *The Pathol. and Diagn. of Diseases of the Chest*, 4th ed., 1840, p. 320.

[2] *Loc. cit.*, p. 322.

[3] *Loc. cit.*, p. 331.

[4] *Traité d'Anatom. et de Physiol. du Système Nerveux*, 1842, t. ii., p. 289.

Volckmann[1] could not, from direct inspection, derive any evidence of the bronchial contractility nor of its dependence on the vagus. But, by means of a complicated arrangement in his experiments, he saw, that irritation of the nerve was followed by muscular contractions, which took place in jerks. He assumed that they proceeded from the lungs, and consequently felt convinced, that the active movements, of which the organ was evidently capable, must, necessarily, serve some purpose in the economy of life.

The experiments of Knaut[2] served to corroborate the statements of Longet and Volckmann. Loewinsohn,[3] on the contrary, was led to infer that the pneumogastric was, in reality, an inhibitory nerve of the bronchial muscles, so that their contraction could only take place when it was paralysed.

Others, again, had either doubtful or completely negative results. After many failures, Wintrich,[4] at last, saw the contraction of the bronchi; but it seemed to him so feeble that it could not be a cause of dyspnœa. Neither Budd[5] nor Rosenthal[6] were even so far successful.

---

[1] Rud. Wagner, *Handwörterb. d. Physiol.*, Bd. ii., 1864, p. 586.

[2] *De vitali, quæ dicitur, pulmonum contractilitate, nervis vagis irritatis.* Diss. Inaug.: Dorp, 1859, p. 33 *seq.*

[3] *Experimenta de nervi vagi in respirationem vi et effectu.* Diss. Inaug.: Dorp, 1858, p. 41 *seq.*

[4] Virchow's *Handbuch d. speziel. Pathol. u. Therap.*, Bd. v., Abth. 1, pp. 190—201.

[5] *Medico-Chir. Trans.*, 1840, vol. xxii., p. 37.

[6] *Die Athembeweg. u. ihre Beziehung: zum N. Vagus*, 1862, p. 232.

Rosenthal could not satisfy himself, that the lungs received motory fibres from the vagus; for in all his experiments, irritation of both its central and its peripheral portions had no effect whatever upon the volume of the organ when the thorax was opened. Subsequently, however, he accepted[1] the results obtained by trustworthy observers, notwithstanding his own negative experience on the point.

Thereupon, Rügenberg[2] attempted to account for the existing conflict of opinions. He had found that, on irritation of the vagus, the lungs were compressed by the upward movement of the stomach and of the diaphragm —a movement which arose from the contraction of the œsophagus; but that they exhibited no change of volume when detached from the surrounding tissues. He, therefore, held, that the oscillations of the manometer, which really proceeded from compression of the lungs, were, erroneously, attributed to bronchial contractions.

The sources of fallacy, which Rügenberg had pointed out, were now carefully avoided by Paul Bert,[3] who experimented only upon lungs after their removal from the body, and after their complete separation from adjoining structures. At first, the effect was negative, owing, as he subsequently learnt, to their state of inflation; but, on their collapse, they readily contracted upon electrical irritation, applied either directly to them or to the pneumogastric. He urged, however, that those contractions were far too feeble and too slow, to have any active share in the mechanism of respiration, and deprecated the hasty construction upon those experiments of any theory on the nature of Asthma.[1]

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[1] Hermann, *Physiologie*, Bd. iv., Abth. ii., p. 243.

[2] *Ueber d. angebl. Einfluss d. N. Vagi auf die glatten Muskelfasern der Lungen. Stud. d. physiol. Inst. z. Breslau*, 2^e Heft, Leipzig, 1863.

[3] *Leçons sur la Physiol. comparée de la Respir.*: 1870, p. 371 *seq.*

L. Gerlach[2] then ascertained, that the force of the pressure in consequence of the bronchial contraction was very slight; amounting, as a rule, to one of from 3—4 mm. of water, once only reaching to 10 mm.

McGillavry,[3] starting from the idea that, in the usual arrangements of the experiments, the manometer did not accurately indicate all the pressure, which the contraction of the bronchioles was capable of producing, forced air through the lungs, the surface of which had been perforated by a number of pin holes; and thus it was found that its force occasionally came up to 30 mm. of water, but generally only to from 8—14 mm.

There was, thus, abundant evidence of the bronchial contractility. The last link in the chain was now forged and the gap was filled up.

ASTHMA, A SYMPTOM OF PATHOLOGICAL CHANGES.

A few dissentient voices were heard, nevertheless, and objections were raised to the supposed nervous origin of Asthma. Some declared, that their observations in practice did not support that view;[4] others admitted, that there were, indeed, cases of paroxysmal dyspnœa of which the nature was obscure; but they saw therein no cogent reason to assume a derangement of innervation. They urged, that there were many and palpable pathological processes which, though capable of producing those clinical phenomena, were still inaccessible to physical diagnosis.[1]

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[1] *Op. cit.*, p. 381.

[2] Pflüger's *Archiv.*, Bd. xiii., 1876.

[3] *Arch. Neerland. des Sciences Natur.*, xii., No. 5, 1877.

[4] George Hirsch, *Spinal Irritation*, 1843, p. 248.

Beau[2] and his pupil Crozant[3] looked upon Asthma as a symptom of a peculiar form of bronchitis. The *râles vibrants*, which were heard during the paroxysm, clearly showed, that the dyspnœa was due to a transient obstruction of the bronchi by means of tenacious inflammatory products.

Some, again, identified it with disease of the heart or of the large vessels;[4] still others with emphysema of the lungs.[5] All from their point of view thought, that those experiments had no bearing upon the pathology of Asthma. Beau even denied that the bronchial contractility had been established.

But those objections, it appears, were not considered sufficiently weighty, and, therefore, received but little attention.

THEORY OF SPASM AND PARALYSIS OF THE BRONCHI.

It was otherwise with the great majority of pathologists. Scarcely had those experiments been completed, or had reports of them emanated from the laboratory, when, immediately, they were seized upon to account for the one obscure form of dyspnœa. In the eager desire to arrive at a solution of the question, sight was lost of the fact, that the results of physiological research had only a limited value—one entirely contingent upon the special conditions, in which they were obtained. At once, they became the base of a number of theories, the defects of which, however, were so conspicuous, that they were refuted, as soon as they were advanced.

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[1] Benvenisti, *Commentario sull' Asma*, 1859.

[2] *Traité experimentale et clinique de l'Auscultat:* 1856, p. 132 *seq*.

[3] *De l'Asthme*, 1857.

[4] Bouilleaud, *Sunt-ne Asthma et angina pectoris symptomatica? Sunt-ne essentialia? Thèse de Concours*, 1826.—Rostan, *Mémoire sur l'Asthme des Vieillards: Journal de Méd.*, 1818, and *Gaz. des Hôpit.*, 1856.

[5] Louis, *Mém. de la Société Méd. d'Observ.*, tom. i., 1836.—Villemin, *Arch. génér.*, 1866.—Virchow, *Deutsch. Klin.*, 1860.

At first, a plus and a minus state of bronchial contractility were each supposed to produce a special form of Asthma.[1] The distinctive characteristics of the one—the spasmodic—were said to be: unusual collapse of the lungs within the thoracic cavity, drawing in of the parietes, and dull sound on percussion; while the physical signs of the other—the paralytic—were stated to be those of emphysema: distension of the chest, hyper-resonance, and prolonged expiration.

The theory of paralysis, however, found but few adherents.[2] Bergson [3] urged against it, that section of the pneumogastric never gave rise to dyspnœa, when the animals were kept at rest. But his chief objection to it was, that it was difficult to imagine a paralysis, which suddenly came and went, as it ought to have done consistently with the origin and subsidence of a dyspnœal paroxysm.

THEORY OF BRONCHIAL SPASM.

The general bias was towards the spasm. Some pathologists went even so far, as to suppose, that the feeling of tightness, which the patients perceived across the chest, positively pointed to a constriction of the air tubes.[1] The great majority of them, however, relied mainly on the physical signs during the attacks. There was undeniable evidence that the air passages were impervious; and as Asthma was presumably a nervous affection, the stenosis could arise in one way only.

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[1] Williams, *Op. cit.*, p. 91, *seq.*

[2] Romberg, *Lehrbuch d. Nerven Krankheiten*, 1851.—Walshe, *Diseases of the Lungs*, 1871, p. 551.

[3] *Op. cit.*, p. 81 *seq.*

The zealous advocacy of Hyde Salter, considerably, helped to foster that view, notwithstanding "his preconceived notions of the pneumogastric pathology of the disease,"[2] with which, as he himself admitted, he had entered upon the question.

Starting with the definition, that Bronchial Asthma was distinguished by an unusually prolonged expiration and by the presence of sibilant rhonchi,[3] he maintained that a combination of such signs invariably indicated the nervous origin of the dyspnœa, whatever else may be present at the time.[4] "Pure Asthma" was, in his opinion, very rare; for, if at all severe, it could not long exist without leading to organic mischief. It was therefore generally observed in connection with anatomical lesions, which were either accidental complications or sequelæ. Indeed, "the most dilated bronchi that he had ever seen,"[5] occurred in one case of Asthma, in which the tendency to the spasm, nevertheless, persisted.

Anxious to prove not only the nervous nature of the disease, but more especially the existence of a bronchial spasm, Hyde Salter unwarily fell into the many errors to which such attempts are apt to lead. To appreciate, however, the difficulties, with which he was confronted, it may be as well to examine, for a moment, the nature of the task, which he proposed to himself to accomplish.

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[1] Hyde Salter, *On Asthma*, 1868, p. 33.

[2] *Op. cit.*, p. 256.

[3] *Op. cit.*, p. 78 *seq.*

[4] *Clinical: Lectures on Diseases of the Chest*, *Lancet*, 1870, vol. i., p. 147.

[5] *Op. cit.*, p. 157.

At best, the available data are few and ambiguous, owing to the circumstances, in which the disease is generally seen. Bronchial Asthma is not, as a rule, the subject of what, may properly be called, clinical observation. Even busy practitioners have, rarely, opportunities of continuously watching, for any length of time, a number of cases; but the fragmentary knowledge of them, which is commonly obtainable, is not conducive to adequate conceptions of pathology.

The fact is, asthmatics are not disposed to consider themselves as patients. Their suffering is forgotten as soon as it is over; and their subjective freedom of respiration in the intervals between the paroxysms gives them the erroneous impression, that nothing can be seriously amiss.

Moreover, the belief has been fostered among them, that the cause of their affliction is but a transient and inscrutable derangement of the nervous system; and that, with the relief of the dyspnœa, there is an end also of the disease. There is even the popular notion, that Asthma affords a lease of a long life.

Hence, as soon as it dawns upon the patients, that they suffer from Asthma, their whole object is, to provide themselves with some remedy, to which they can have recourse in their hour of need. Thus, advice never is and for obvious reasons never can be, sought in the mild and short attacks. It is only in the severe and protracted paroxysms, when their talisman fails them, that they look for help elsewhere. But then, in the presence of the terrible suffering, the actual condition is such as to render an exhaustive examination impracticable. Indeed, the uppermost thought in the mind of the practitioner is, to bring speedy relief, notwithstanding all dubious points of pathology, rather than lose one moment in, what may seem to be, superfluous inquiry. All that he has to go by, are the signs of an obstruction of the air passages; but there are, at the time, no means of deciding on its nature. To infer the cause of the dyspnœa from the striking effect of the treatment would be, here, as inadmissible, as, under similar conditions, in a case of pneumonia or of pleurisy.

The great majority of asthmatics present themselves in the intervals between their attacks, when they, with justice, declare, that they are seen at their best. At the time, the physical signs may be either altogether negative, or so slight as to be deliberately disregarded as "insignificant." If there is evidence of abiding structural lesions, the actual freedom of respiration proves, that they cannot be the cause of the dyspnœa. Anamnesis gives equally unsatisfactory results. Of the origin of the disease, the patients and their friends are, almost always, unable to make definite statements. On account of its insidiousness, it has either never been observed, or has been forgotten, in the lapse of time. Some one or the other is said to have made, at one time or the other, the diagnosis of Asthma. Thus, there only remain the circumstantial accounts relating to the fitful appearance of the paroxyms in the midst of perfect health; to their trivial and incomprehensible causes; and to the success or the failure of certain remedies.

It will be readily conceded, however, that the statements made by patients—no doubt in good faith and with all sincerity—have, here, no greater value than is usually attached to them. It is not in the nature of things, that the principals in suffering, or the interested witnesses of it, could, correctly and impartially, observe and record facts. Their judgment is necessarily biassed by their feelings, and here, in addition, not infrequently so, by popular theories, which please their fancies. Not that their reports should be altogether rejected: they, no doubt, have their value; only they are unsuitable to build a theory of pathology upon, unless after being carefully sifted.

As a rule, such sifting is impossible, for those reports refer to the past. If, for instance, a patient declares that, whilst in perfect health, he was seized with an attack of Asthma, on visiting a house surrounded with ivy, it is certain, that he had dyspnœa. But there is no means of ascertaining that, notwithstanding his alleged freedom of breathing on entering the house, his chest was free from pathological changes, nor that the ivy was the cause of his suffering. Such a statement—only one of thousands, upon which the present pathology of Asthma rests—must stand on its own merits. It does not warrant, however, the conclusion, that the *genius loci*, determined by the ivy, produced, in an otherwise healthy individual, an attack of dyspnœa; nor that this was due to a disturbance of innervation. Nothing, on the other hand, is more certain than that, when the opportunities are given of continuously observing a number of cases so as to be able to verify those statements, much, which is reputed fact, is found to be only dogmatic assertion; much, which is really so, is by no means as absolute as alleged.

Heedless of the sources of fallacy, by which he was surrounded, Hyde Salter accepted the mass of stories, that were told him on the origin and cessation of the dyspnœa, although he admitted, that they bordered on romance.[1] To weld them, however, into a homogeneous whole, he endowed the disease with a "double capriciousness": the one peculiar to "the disease in general"; the other, to "each case in particular." From that capriciousness, again, and from the physical signs, he inferred, without hesitation, the nature of Asthma.

Nothing, however, more plainly shows the value of his theory, than the practical lesson which he himself had learnt from it, and which he was anxious to enforce: that it was desirable, "if a case was going on ill, to make a change, *any* change, no matter whether the object was very definite or the therapeutics very rational"; "for the most blind and purposeless treatment was often attended by the best results."[2]

The views of Hyde Salter are fully shared in by Biermer.[3] He, too, sees in the efficacy of a dose of chloral, a mathematical demonstration that the disease is nervous. He supposes, that a spasm of the bronchi interferes with respiration, just as colic of the intestines does with the onward movement of their contents. But this analogy is inadmissible, because the intestines are not hermetically sealed to the walls of a cavity, which is constantly enlarged by outward traction. To the objections, which had meanwhile been raised, that the physical signs, during the paroxysm, do not correspond to those, which ought to follow from a bronchial spasm, he replies, that while the forcible inspiration overcomes the constriction of the air-tubes and distends the alveoli, the forcible expiration compresses the bronchi and prevents the escape of the air. This valvular action would be imaginable, if the expiratory forces and the bronchial muscles were antagonistic; whereas the former, essentially, reside in the latter.

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[1] *Op. cit.*, p. 291.

[2] *Op. cit.*, p. 104.

[3] Volkmann's *Sammlung klin. Vorträge*, No. xii.

SPASM FROM HUMORAL CHANGES.

While most practitioners considered Asthma a substantive disease, there were some, who looked upon the bronchial spasm as a general pathological process and a part expression of other affections.

Thus Duclos[1] assumed that the blood contained an herpetic principle, which, alternately, exerted its specific influence upon the skin and upon the mucous-membrane of the bronchi. The irritation of the latter induced spasm and dyspnœa. Similar was the origin of Asthma supposed to be, when the special "materia peccans" of gout,[2] rheumatism, and hæmorrhoids[3] was eliminated by the lungs.

[1] *Bulletin général de Therap.*, Ap., 1801.

[2] Murchison, *Lectures on Functional Derangement of the Liver.*

[3] Trousseau, *Clinique Médicale de l'Hôtel Dieu.* 1868. Tom. ii., p. 453.

SPASM FROM BRONCHIAL EXUDATION.

Amidst the speculations on Asthma the observation of asthmatics was, apparently, neglected. No doubt, the physical signs received, at all times, when opportunities occurred, their full share of attention; not so, however, the associated phenomena, by which alone their nature could, rightly, be interpreted. No notice was taken of the only other objective sign—the sputum, with which the paroxysm invariably terminates; although, as a pathological product, it must afford some clue to the pathological process. From the peculiar point of view, from which the disease was regarded, such examination seemed superfluous; for the expectoration could only be a consequence, or possibly an exciting cause, of the spasm.

No importance was, therefore, attached to the observations of Lefèvre. Himself[1] asthmatic, he had found, in his own case and in that of an asthmatic friend of his, that the relief of the dyspnœa generally coincided with the expectoration of a peculiar kind of sputum. He described this as being "of a grey colour, tenacious or firm in consistency; filamentous in shape, like boiled macaroni, or twisted into spirals; and moulded upon the ultimate ramifications of the bronchi, in which it had probably stagnated for a time." He had no doubt, that the proximate cause of the dyspnœa was a bronchial spasm; but the essence of the disease was to him a peculiar inflammatory process, which caused the increased irritability of the bronchial muscles, and gave rise to those peculiar inflammatory products.

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[1] *Recherches sur l'Asthme: Mem. couronné par la Soc. de Méd. de Toulouse. Journ. Hebdom.*, 1835.

Subsequently, Biermer[1] mentioned, that such spiral formations were of frequent occurrence in croupous bronchitis; and he significantly added, that cases of that kind may be readily mistaken for Asthma, unless the diagnostic sputum be detected.

The exudation of Lefèvre and croupous bronchitis being evidently identical processes, the idea was inevitable, that either Asthma was a symptom of the latter, or, if they were different affections, that the diagnosis could be established only upon the presence or absence of the characteristic casts.

The matter, however, remained in abeyance, till Leyden[2] on microscopic examination of the sputum in Asthma, found "tubular contorted bodies with obliquely spiral lines and a narrow homogeneous-looking thread, which ran along their axis and terminated with a spiral twist."[3] At the same time, he observed a peculiar form of octahedral crystals, which had already been seen by Charcot and Robin, by Förster and Friedreich, in cases of bronchitis and of "catarrh sec." At first, those crystals were supposed to be a mucine compound, but subsequently they were found to consist of a peculiar organic base.[4] But they had been observed also in the normal and the leukæmic bone-marrow and in the sperm; and Leyden, therefore, concluded, that they were derivatives of disintegrating white blood-corpuscles.

As regards the pathogeny of Asthma, Leyden attached less importance to the presence of those spiral structures than to that of the crystals. While conceding the close resemblance, if not the identity, between it and croupous bronchitis, he thought, that the sharp points of the crystals irritated the terminal filaments of the vagus in the bronchial mucous membrane, and thus produced a bronchial spasm.

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[1] Virchow's *Handbuch d. Spez. Pathol. u. Therap.*, Bd. i., p. 720.

[2] *ZurKenntniss d. Bronchial Asthma: Virch. Arch.*, vol. liv., 1872.

[3] *Loc. cit.*, p. 349.

[4] Schreiner, *Liebig's Annal.*, Bd. 194, p. 68.

The facts to which he had called attention were soon confirmed on all hands. Only Zenker[1] and Merkel[2] urged, that crystals occurred also in the cases in which, at the time, no paroxysmal dyspnœa existed.

Ungar,[3] on the other hand, pleaded, that the dyspnœa was determined by those spiral formations, rather than by the crystals; and that the assumption of a bronchial spasm was unnecessary and gratuitous. He held, that the Asthma arose from obstruction of the bronchioles; and explained, that the physical signs during the paroxysm were due to the fact, that the forcible inspiration allowed the entrance of air past existing plugs, which, however, impeded its escape. This view is evidently untenable; for even if the process were as diffuse as that of an ordinary form of capillary bronchitis, it would not account for the asthmatic type of the dyspnœa; and there are many instances, in which the paroxysm subsides on the expectoration of but a few pellets, while neither immediately afterwards, nor later on, are there any signs of their continued presence in the air-passages.

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[1] *Tageblatt d.* 45 *Versamml. deutsch. Naturf. u. Aerzte:* Leipzig, 1872.

[2] Ziemssen's *Handbuch*, vol. i.

[3] *Verhandl. d. Congr. f. Innere Mediz.*, 1882. *Arch. f. klin. Mediz.*, Bd. xxi., Heft 4.—*Sitzungsber. d. Niederrhein Gesellsch. f. Natur. u. Heilk.*, 1881, Feb. 21st, and Dec. 19th, March 1st, 1881.

The impulse had thus been given to microscopic examination of the sputa. Curschmann,[1] then, saw Leyden's spirals in a large number of cases of Asthma. He thought, as Lefèvre had done before him, that they were the product of a peculiar form of bronchitis—"bronchiolitis exudativa," causing dyspnœa by reflex irritation. In his opinion, Asthma is, essentially, a neurosis of the vagus. He concedes, as possible, a primary form of central origin; but he does so only out of deference to an established theory, not upon actual observations. As a rule, Asthma is a secondary affection and a bronchial spasm. Such a spasm may be induced by the irritation of any part of the body, but more especially by that of the different sections of the respiratory tract; and, here, the most frequent cause of it is the presence of those spirals. It was, soon, however, pointed out, that these are found in the sputum, when no dyspnœa exists.[2]

THEORY OF SPASM OF THE DIAPHRAGM.

Early in the existing controversy, there were those who, while admitting, as an indisputable fact, the primary nervous origin of Asthma, yet contended, that the supposed bronchial spasm was for many reasons impossible, and, more especially, inconsistent with the physical signs during the paroxysm.

Budd who, as already mentioned,[3] could not, by his own experiments, satisfy himself of the very existence of a bronchial contractility, thought, that Asthma was, in all probability, due to certain morbid changes in the blood;—changes which induced a spasm of the diaphragm and of the other muscles of respiration.

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[1] *Deutsch. Arch. f. klin. Mediz.*, Bd. xxxii.—*Verhandl. d. Congr. f. Inn. Mediz.*, 1885.

[2] *Vierordt. Berl. klin. Wochensch.*, 1883, No. 29.—*v. Jacksch. Centralb. f. klin. Med.* 1883, No. 31.—Vincenzo, *Annal. Univers. di Med.*, vol. cclxvii.—*Pel. Zeitsch. f. klin. Med.* Bd. ix., Heft 1, 1885.

[3] *Vide* p. 13.

A similar view was adopted by Wintrich and others.[1] Without denying the bronchial muscles all participation in the production of the dyspnœal paroxysms, Wintrich held that their share in it would be slight, but that probably a spasm of the glottis was concerned in them.

But, hitherto, a tetanus of the diaphragm had not yet been observed in man, and was, therefore, only a hypothesis. In animals, Duchenne[2] had frequently produced it by faradisation of the phrenic nerves. The experiment showed, unmistakably, some of the symptoms, peculiar to Asthma:—extreme dyspnœa, distension of the lower half of the thorax, projection of the epigastrium, and diminished frequency of respiration. But herein the experiment differed from Asthma: that *death invariably ensued, when the irritation of the phrenic was continued for more than a few minutes.*[3]

Valette, however, subsequently met with, and reported, a case of acute articular rheumatism, in which a fatal attack of dyspnœa had lasted for *several days uninterruptedly*; and from the symptoms of which he, distinctly, recognised a tetanus of the diaphragm. Its occurrence in man being thus established, Duchenne concluded, that it was the proximate cause of asthmatic paroxysms.[4]

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[1] Virchow's *Handb. d. speziel. Pathol. u. Therap.*, Bd. v., Abth. 1, 1885, pp. 190—201.—Lehmann on "Pathogenesen of Asthma," *Bibl. für Laeger.*, 1866; *Virch. Jahresb.*, 1866, vol. ii., p. 103.—Kidd, *Dublin Quarterly Jour. of Med. Science*, 1861.

[2] *De l'Electrisation localisée*, 1855.

[3] *Op. cit.*, p. 459.

[4] *Op. cit.*, 2nd edit., 1861, p. 917.

Bamberger[1] accepted this doctrine. He, too, had seen, in an otherwise healthy labourer, a fatal case of tetanus, attended by extreme dyspnœa. Tetanus of the diaphragm may, in his opinion, be of central and of reflex origin, the latter generally proceeding from emphysema, bronchitis, pleuritic effusion and tuberculosis. One form of Asthma, he maintained, was probably caused also by paralysis of the diaphragm; while the so-called hysterical Asthma — a dyspnœa of an inspiratory type — hitherto erroneously attributed to spasm of the glottis, was, in reality, due to clonic contractions of the muscles of respiration.

Neither Lebert[2] nor Jaccoud[3] were convinced, that a bronchial spasm or a tetanus of the diaphragm satisfactorily explained a paroxysm of Asthma. Both these processes were, in their opinions, concerned in its production.

Traube,[4] and later on Fräntzel,[5] conceded, that the diaphragm was firmly contracted during the paroxysm; but both held, that such contraction was merely a part of an altered mechanism of respiration, consequent upon the presence of an obstacle in the air passages. At first Traube supposed, that the pulmonary tissue was the seat of peculiar degenerative changes which predisposed to a congestion of the mucous membrane—a "catarrhus acutissimus,"—and thus led to a rapid occlusion of the bronchial tubes. Subsequently, however, he[1] was inclined to admit, that the congestion might, possibly, be of vaso-motor origin.[2]

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[1] *Ueber Asthma Nervosum.—Würzburg mediz. Zeitsch.*, Bd. vi., 1868, pp. 102—116.

[2] *Klinik d. Brustkrankheiten*, 1873, Bd. v., p. 439 *seq.*

[3] *Traité de Pathologie interne*, 1869, t. i., p. 810 *seq.*

[4] *Deutsche Klinik*, 1860, p. 467.

[5] *Charité Annal.*, 1877, p. 294.

Germain Sée[3] maintained, that Asthma was a neurosis of the vagus, accompanied, but never produced, by organic lesions, and consisted of three elements: bronchial exudation, tetanus of the diaphragm, and emphysema. The former two elements are said to be "essential to the disease"; the latter, emphysema, is considered to be only a complication, but one of so frequent occurrence as to become an almost integral part of the disease.

This theory is, in its main points, adopted by Riegel,[4] who, however, insists, that the diagnosis of nervous Asthma is dependent on the absence of such organic lesions as would, otherwise, satisfactorily account for the origin of the intense dyspnœa.

By a series of experiments, which he performed together with Edinger,[5] he was able to confirm all that had, hitherto, been known and accepted, on the physiology of the bronchial muscles, and on that of the vagus. He endorses the objections, which Bamberger had raised to the theory of a bronchial spasm, without, however, entirely rejecting it; and believes that this, together with vaso-motor disturbances of the bronchial mucous membrane, explains the sibilant rhonchi, which are heard during the paroxysm.

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[1] *Gesammelte Abhandlungen*, Bd. iii., p. 167.

[2] *Vide* also Sir Andrew Clark. "Observations on the Theory of Bronchial Asthma, viewed in the light of the Pathology of Hay Fever." *American Journ. of Med. Science*, Jan., 1886.

[3] *Diction. de Médec. et de Chirurg. pratiques*, t. iii., 1865, p. 585 *seq.*

[4] *Verhandlung. d. Congress. f. Inn. Med.*, 1885.

[5] *Experiment. Untersuch. z. Lehre v. Asthma: Zeitsch. f. klin. Med.*, Bd. v., p. 413 *seq.*

But, such spasm of the diaphragm finds support, neither from physiological data, nor from clinical observation.

For, in the first place, the movements of the diaphragm, on irritation of the inspiratory fibres of the vagus, bear no resemblance to the presumed spasm. Notwithstanding its descent, there are slight, but regular breathing excursions.

Secondly, a spasm could not exist for any length of time, without endangering life. Death actually took place in the cases reported by Valette and Bamberger.[1] Yet, attacks of Asthma may last for days uninterruptedly, and do end in recovery.

Again, the spasm would have no influence upon the antero-posterior, nor on the transverse, diameters of the thorax; whereas both these are enlarged during the paroxysm.

And lastly, in most cases of Asthma, the action of the diaphragm is of an inverse type: it ascends on inspiration, the epigastrium being retracted, and descends on expiration; while the base of the thorax is distended, causing bulging of the abdomen.

THEORY OF NASAL ASTHMA.

A casual observation by Voltolini,[2] henceforth, directed attention to affections of the nose as being presumably the starting-point of Bronchial Asthma. He reported a case, in which the disease was cured by the removal of a polypus;[3] but he, expressly, added, that a successful issue of the operation may be expected only in specially favourable circumstances.

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[1] *Vide* pp. 28 and 29.

[2] *Die Anwendung der Galvanokaustik, etc.* 2^e Aufl., 1871, p. 247.

[3] *Op. cit.*, p. 312.

Haehnisch,[1] whose own observation fully confirmed the usefulness of that operation, explained, that the pressure of the polypus upon the nervous fibres within the naso-pharyngeal cavity, caused, by way of reflex, a spasm of the bronchi. It was necessary, however, for such a result to occur, that the lungs should be specially predisposed to it; for, not unfrequently, a polypus existed, without producing that effect. But, as a rule, the growth itself was sufficient, by its disturbing influence upon respiration, to render the lungs a "locus minoris resistentiæ," and thus to lead to asthmatic paroxysms.

Weber[2] observed, that attacks of dyspnœa, were frequently ushered in by acute coryza, which was, apparently, not due to mechanical or chemical irritation. He, therefore, concluded that the congestion arose from vaso-motor disturbances; and that the process extended from the nose to the ultimate ramification of the bronchi.

Störk[3] examined, by means of the laryngoscope, a patient during a paroxysm of dyspnœa, and could thus verify the existence of a tracheal hyperæmia. But he held that a spasm of the diaphragm was equally concerned in its production.

Nothing, however, is known, as yet, of a congestion of the air-passages from nervous disturbances. On the contrary, the progress of the disease, so far as clinical observation goes, is entirely opposed to such hypothesis. Sneezing fits and swelling of the nasal mucous membrane, indeed, precede the appearance of Asthma; but they do so, often, for days. In the interval, there are unmistakable signs of hyperæmia of the bronchi, yet there is *no* dyspnœa, when the patients are at rest.

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[1] *Zur Aetiologie u. Therapie des A.-bronchiale.—Berl. kl. Wochensch.*, No. 40, 1874.

[2] *Tageblatt d.* 45ten *Versammlung deutsch. Naturf. u. Aerzte*, 1872—Haring, *Ueber Bronchial-Asthma.* D. I. Halle, 1873.—Moritz, *Ueber Bronchial-Asthma*, 1884.

[3] *Mittheilung. üb. Asth. Bronch. u. die mechan. Lungenbeh.*, 1875.

Hack[1] opportunely warns against enthusiastic exaggerations, although he admits the alleged origin of Asthma from nasal affections, and the possibility of usefully influencing it by their surgical treatment. The facts, however, are that in many cases of nasal polypi, there is no Asthma; that in some, in which both are associated, the dyspnœa may subside for years, while the polypus is left undisturbed; and that, in others, the latter may be effectually removed, whereas Asthma persists or returns with its habitual severity.

Nevertheless, the theory is alleged to find support in the rapid cures which are said to be effected, in Asthma, by the removal of a polypus, and by galvano-cautery of the hypertrophied cavernous tissue of the nose.[2] The results thus obtained are held[3] to afford sufficient evidence, that the dyspnœa proceeds from irritation of the Schneiderian membrane, and that such irritation leads to reflex contraction of the bronchial muscles.

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[1] *Ueb. d. chirurg. Behandl. Asthmatischer Zustände; Verhandl. d. Congress. f. Inn. Mediz,* 1885.

[2] Shäffer, *Asthma u. seine Behandlung: Deutsche Mediz. Wochenschrift,* Nos. 32, 33, 1879.—B. Fränkel, *Ueber den Zusammenhang von A. nervosum u. Krankheiten d. Nase: Berl. Kl. Wochensch.,* Nos. 16, 17, 1881.—Bresgen in *Volkman's Sammlung Klin. Vort.,* No. 216.

[3] *Vide* also discussion in the *Congress. f. Inn. Med.,* 1885.—Schnitzler, *Wiener Med. Presse,* 1883.

CHAPTER II.

CLINICAL HISTORY OF BRONCHIAL ASTHMA.

Origin of the Disease—Forms of the Disease: The Acute; the Chronic—Description of the Paroxysms: Disturbances of the Nervous System, of Secretion, of the Digestive Organs, of Circulation; Physical Signs on the part of the Lungs—Sputa—Symptoms of the Intervals—Duration of the Paroxysms—Time and Mode of Access—Frequency—Alleged Periodicity—Premonitory Signs—Alleged Capriciousness—Alleged Alternations with other Diseases.

ORIGIN OF THE DISEASE.

OF the manner in which Asthma originates, nothing is positively known. The literature on the subject contains no reference to that particular point; and opportunities of observing it are, evidently, very rare.

In the routine of practice, the patients are seen, only when they have already been "asthmatic" for a considerable time. From the reports, which they or their friends furnish, it is certain that no one, in a state of perfect health, becomes suddenly, and without some warning, subject to the peculiar attacks of dyspnœa. The sole exceptions to this rule are perhaps the isolated instances, in which foreign bodies have unperceivedly entered the air-passages.[1]

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[1] The most remarkable instance of the kind is that of a Dr. Nooth, who had for fifteen years suffered from spasmodic Asthma

In its typical form, however, the disease, before it assumes its distinctive characteristics, is invariably, and for an indefinite period, preceded by a series of symptoms which constitute one of its integral parts.

In the vast majority of cases, all present troubles are assigned to what appears to be a peculiar form of inflammation, which starts in the pharynx, rapidly spreads upwards along the contiguous mucous membrane, but is, at first, effectively arrested in its downward course by the projecting tissues of the larynx. The irritation which it produces—chiefly severe fits of sneezing and cough—is supposed to be a mere symptom of an "ordinary cold"; or, if it occur in the height of the summer, when such causation is obviously inadmissible, one of "hay fever." But because a "cold" is, presumably, still within the limits of health, and because "hay fever" occurs, after all, only once a year, the discomfort, which arises from them, receives but little attention; and when at last Asthma is fully developed, it appears to have come on quite insidiously.

Accordingly, many patients declare—and seemingly with good grounds—that before the onset of their dyspnœal paroxysms, they never had a day's illness. On inquiry, however, it will be found that, not only were those symptoms present, but they were unusually severe and protracted; and though they escaped the notice of the sufferer, they did not so escape the notice of his friends.

Thus, one asserted that he had always enjoyed good health, and did not know what illness was, till he became asthmatic. But it was soon elicited from him, that

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till he expectorated a shot.—*Transactions of the Society for the Improvement of Medical Knowledge*, vol. iii.

while attending his brother's funeral, his relatives were struck by his distressing cough, and entreated him to seek advice for it. Shortly afterwards the dyspnœa commenced. Another, who was equally emphatic on the same point, was reminded by his wife that he had, for a long time, been subject to a form of cough, which sounded to her like whooping cough, and had greatly disturbed his nights' rest.

In many instances, especially in children, the dyspnœal paroxysms are traced to some inflammatory affection of the chest, which was either idiopathic, or a part expression of measles and whooping cough. The stories run, that recovery was greatly delayed; it was particularly noticed, that the cough had changed its character: painful and severe at first, it succeeded in clearing the chest of a very viscid exudation; but subsequently, though its frequency had been greatly reduced, yet its efficacy was perceptibly diminished. The expectoration, it is said, required an unusual effort, during which the breathing became visibly embarrassed; such paroxysms occurred chiefly at night, and returned with great obstinacy, whereas the days were comparatively free from them; gradually, however, the nocturnal attacks also subsided, and there was some prospect of a favourable termination of the disease.

Apparently, the general condition, which had been greatly impaired, showed no signs of improvement. The respiration also became readily oppressed on the slightest exertion, or by excitement. Whatever hope there was of a recovery, was soon altogether destroyed. After an interval of days, or even of weeks, during which all symptoms had considerably abated, the dyspnœa returned, with as great a violence as ever, from unknown or inappreciable causes.

It now began to "dawn" upon the patient and his medical adviser that, whatever may have been the original diagnosis, the disease which then presented itself was Asthma.

FORMS OF THE DISEASE.

In the habitual asthmatics, such as they are commonly met with, the disease is seen in two forms.

The Acute.

Its mode of access is that of an acute disease. As a rule, it is ushered in by shivering or a distinct rigor—the "cold" to which patients trace their dyspnœa. The temperature rises, and generally fluctuates between 101° and 104° Fahr., rarely exceeding the latter. At the same time, there are signs of constitutional disturbances, such as, general malaise, frontal headache, weariness and aching of the limbs, and incapacity for mental and bodily exertion. Intense pain between the shoulder-blades is early complained of, and, occasionally, all the muscles of the chest feel tender.

The first local symptom is itching or a burning sensation, "as if due to the presence of cayenne pepper," on the nasal surface of the soft palate. The irritation soon extends to the internal ears, to the nose, to the roof of the mouth, and to the root of the tongue. Frequently, also, the eyes are affected, and there is a feeling of pressure, as if grains of sand were in them. Then, perhaps in a few hours, oftener in the course of a day or so, follow violent fits of sneezing, distressing cough, and oppression at the chest.

On the first appearance of the constitutional disturbance, the mucous membrane of the pharynx is deeply red and swollen. The hyperæmia, with its copious transudation, rapidly spreads upwards to the nose and conjunctivæ; and now that the barrier has been overcome, also downwards to the larynx and bronchi. Extensive collateral œdema accompanies those inflammatory changes; the eyelids are baggy, and, as a rule, the face is puffy, and smarts. The regional lymphatic glands, often as far as the middle of the neck, participate in the process, and are considerably enlarged.

In most cases there are, at the same time, various forms of cutaneous eruptions. Occasionally, there are herpes vesicles on the lips, or the whole face may be covered by them. Not unfrequently, a few isolated and dark blue papules, probably *erythema papulatum*, appear at, or near the chin, and on one side of the neck, and cause a great deal of itching. In two instances, there was urticaria all over the body; in two others, one wheal only occurred on one arm, and at the angle of the right scapula. Once, spots of erythema were seen on the chest and on the extremities. In a large number of them, the alleged irritation of the skin develops, later on, into an attack of acute eczema.

The inflammatory process is subject to variations in extent and intensity. After frequent recurrences of the disease, it is chiefly localised in the air passages from the trachea downwards, while the upper portion of the respiratory tract is but slightly affected. There are always, however, symptoms on the part of the pharyngeal and nasal mucous membranes, so that sneezing or running from the nose is hardly ever absent.

Often, the hyperæmia is attended by a croupous exudation. Layers of pseudo-membranes are then seen on the pharynx, on the tonsils and on the uvula; while the tongue, especially near its root, is thickly coated by them. Parenchymatous tonsillitis is frequently observed in children.

The implication of the larynx occasionally gives rise to a peculiar kind of cough which may, readily, be mistaken for pertussis, if this happens to be prevalent. In one case, the daughter of an asthmatic lady was sent home from school, where such epidemic had just broken out. In due course, both mother and daughter showed what appeared to be indubitable signs of the infection, although the former declared, that she had had several attacks of whooping cough. It was subsequently found, however, that with each recurrence of her Asthma, which took place twice or three times a year, her cough always presented the same character.

This series of symptoms persists for several days, during which, notwithstanding the cough and the oppression at the chest, the patients are able to follow their usual avocations —not, however, without some difficulty. Their chief complaint is, that every movement, especially going upstairs, greatly tries their breathing, whereas they are tolerably comfortable when at rest. On examination at the time, there are, in uncomplicated cases, hyper-resonance on percussion, feeble respiration, and the various kinds of rhonchi all over the thorax.

Occasionally, however, the pulmonary tissue is seemingly involved in the peculiar changes, which affect the bronchi. There is, at all events, a marked dulness of some parts of the back of the thorax, thus simulating a lobular, or by the confluence of several lobules, even a lobar pneumonia.

Shortly afterwards, the dyspnœa sets in at night with great severity, but subsides only imperfectly, after a persistence of varying duration, to return once or twice in twenty-four hours. For a week, or even longer, there may be a series of such paroxysms, closely following each other at intervals, during which the respiration continues to be greatly impeded; but at the end of it, recovery is often complete, and both subjective and objective symptoms may, then, entirely disappear.

The Chronic.

The chronic form, by far the more frequent, has evidently developed itself out of the acute. It is mainly distinguished by the constant recurrence of dyspnœal paroxysms, which are generally of short duration, and are sharply separated by intervals of a more or less complete freedom of respiration. But this freedom is entirely subjective. It is due to a reduction of the respiratory wants, by means of which a compensation is established for the exclusion of a part of the organ from its function. For in all such cases, there is evidence of a persistent but imperfect stenosis of several large bronchi. Over some portions of the lungs, the respiratory sounds are either altogether suppressed, or are very feeble, while the resonance remains unimpaired, or is but slightly tympanitic. No definite statement can, however, be made as to the extent of the obstruction, nor as to the comparative frequency of its seat; chiefly because the physical signs readily change place and character from day to day. This much, however, is certain: that the lower lobes are oftener affected than the upper, and the right side is more so than the left. In these circumstances, acute exacerbations present, as a rule, a more asthenic type.

DESCRIPTION OF THE PAROXYSMS.

The slight attacks of Asthma, which may return once or even several times in twenty-four hours, are of too short duration to be ever the object of clinical observation. They are generally described as a feeling of constriction across the chest, or as if a heavy load were weighing on it, and threatening suffocation. But these painful sensations readily subside if, by some means, the cough is rendered sufficiently powerful to expectorate a pellet of mucus.

Nothing, on the other hand, is so distressing to witness, and, no doubt, still more so to endure, as the hard struggle for breath, in the typical paroxysms. The patients rarely complain; in answer to enquiries, they merely point to the sternum, behind which they distinctly perceive an obstacle impeding the access of air. But their difficulty in supplying the necessary amount of air is obvious enough, from the number of the muscles which at the time are engaged upon that task. In case of need even those that only indirectly help in expanding or compressing the chest, may be forcibly contracted. To facilitate their exceptional function certain attitudes are instinctively assumed: the patients either stand or sit erect, grasping some solid object; or they bend forward at a right angle, their heads resting on their knees. These postures are anxiously preserved, for fear lest the slightest movement increase the existing trouble.

Often, an incessant cough torments them, but makes no impression upon the obstacle; and the copious frothy expectoration, which accompanies it, brings no relief. All efforts seem in vain to remove the obstruction. And yet, in spite of this, they succeed in satisfying the most imperious respiratory wants. In some instances, the ventilation of the lungs appears to be perfect. In others, however, where the impediment is considerable and the energy of the heart impaired, there is a great amount of cyanosis. To what extent the interchange of gases is, in these circumstances, carried on, is as yet unknown. Hervieux[1] is said to have found in two analyses, that the oxygen had entirely disappeared from the expired air.

Disturbances of the Nervous System.

The patients are generally fretful. When the supply of air is imperfect and asphyxia seems imminent, they are unavoidably in a state of the greatest terror. The feeling of the supposed "constriction" of the air passages causes them much distress; and it probably accounts for the forcible respiratory movements, which often appear to be out of proportion to the extent of the existing obstruction. From the disturbance of the cerebral circulation there is headache, which is greatly increased by cough. Loss of consciousness has never been observed, although it was reported to have occurred in one case. Marked somnolence and trismus were once seen in a patient, who had, for fifteen years, shown unmistakable signs of granular atrophy of the kidneys, and had been subject to Asthma six years before his death.

The tetanic contractions of the muscles are attended by acute pain. The thorax, distended to its utmost limits, feels as if about to burst. The patients groan and apply their hands to the left hypochondrium and to the cardiac region.

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[1] "Recherches sur l'Emphysème palmonaire."—*Arch. de Méd.*, 1861, t. xvii.

The temperature always rises, even in the chronic form of the disease; and generally fluctuates between 99·5° Fahr. and 104° Fahr.; 101° Fahr. and 103° Fahr. were most frequently noticed. It is, as a rule, lower with copious, and higher with scanty perspiration. In cases of great cyanosis, heat is retained and becomes very oppressive, so that even in the winter, asthmatics prefer to have no fire in their rooms, to keep their windows open, and beg to be cooled by fanning.

Disturbances of Secretion.

The forcible respiratory movements stimulate the secretions of the various organs, especially if the energy of the cardiac muscles remains unimpaired.

The skin is flushed and bathed in perspiration, so that every article of dress, which happens to be worn at the time, is soaked through. From the rapid evaporation of the water, there is a great loss of heat; and as this is abstracted from the body, the patients begin to shiver and ask to be wrapped up. When the obstruction is considerable and the action of the heart feeble, the perspiration is much less copious, and the surface feels cold and clammy.

At the same time, the glands also of the nasal and of the buccal cavities are in a state of increased activity. Occasionally, there is continual dripping from the nose, and the discharge of saliva may be so considerable as to amount to several pints. Probably the incessant and tormenting cough is due to the presence of the saliva and to its irritating effect upon the larynx.

This salivation may simulate œdema of the lungs, the told him. There was loud tracheal rattling, and his whole appearance was that of a dying man.

There was no cough, nor expectoration. Respiration was 30; the thorax distended to its utmost limits; the diaphragm ascended with inspiration and descended with expiration, which was accompanied by bulging of the abdomen. From the apices to the third ribs, resonance was dull, and respiration feeble; there were loud sonorous rhonchi. In the axillary regions, and at the bases in front, the resonance was tympanitic, the respiration loud, but no rhonchus of any kind was heard. The back could not be examined. Over the trachea was loud rattling, which was not conducted downwards.

The pulse was 120, very small and soft. The cardiac area could not be made out. The impulse was feeble; the sounds were indistinct, but louder on the right than on the left.

He soon recovered, and expectorated only a few pellets of a very tenacious, yellowish, and nummular sputum, of characteristic composition. There was no expectoration of serous or of frothy fluid. The next day the dulness at the upper lobes began to clear up, and entirely disappeared in three days.

Whether the secretion of the glands in the bronchial mucous membrane is equally increased, is difficult to say. Towards the end of the attacks, there are generally signs of a certain amount of fluid in the air tubes; but it is possible, that this may have a mechanical, as well as a functional, origin. For, owing to the distension of the thorax, the air behind an imperfect obstruction of a bronchus becomes rarefied, and thus a true "*hydrops ex vacuo*" may develop itself.

Profuse diuresis is always present. The urine is limpid, and, according to an analysis by Dr. Ringer,[1] deficient in urea and in chloride of sodium. In experimental dyspnœa, the urea is said to be increased;[2] but clinical observations on this point are still wanting. In cases of renal disease, the albumen, generally, increases at the time; traces of it were detected in several cases, in which there were, neither before nor after the paroxysms, signs of kidney mischief.

Disturbances of the Digestive Organs.

The appetite completely fails. However long the attack may last, the patients remain without food. They have not breath enough to eat, and are afraid that anything solid may increase the dyspnœa. There is, however, increased thirst; the mouth feels dry and parched, owing to the breathing being carried on through it, and to the considerable loss of water by the copious perspiration.

A peculiar odour, somewhat like that produced in singeing poultry, emanates from the mouth. It is especially marked in the acute form of the disease, and is perfectly identical in all cases. The tongue is generally thickly coated.

The breathing through the mouth leads to the sucking of air; so that the stomach becomes greatly distended, and causes much discomfort. Eructation then greatly relieves.

There are obstinate constipation, a feeling of nausea, and often a tendency to vomiting. The slightest thing is apt to produce sickness, which is often accompanied by faintness. Probably the emesis is due to the mucus, which sticks to the pharyngeal wall.

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[1] *Parkes on Urine*, p. 319.

[2] Salkowski u. Leube, *Die Lehre vom Harn.*, 1882, p. 471 *seq.*

Disturbances of Circulation.

As a rule, the cardiac area cannot be made out. According to the state of nutrition of the cardiac muscles, the impulse may be either distinct or imperceptible. There is almost always epigastric pulsation. The cardiac sounds are generally feeble, much louder on the right than on the left, showing the tension in the pulmonary artery, the sounds of which are occasionally accentuated. Palpitation is often complained of.

The frequency of the pulse varies. It is seldom less than 90, and generally rises to 120 in the minute. The arterial tension is, as a rule, diminished, and the wave low. In some cases, the pulse is very small, and almost empty; and in others, again, it is distinctly paradoxical—*i.e.*, during inspiration there is a very marked reduction both in the tension of the vessel and in the height of the wave. There may be distinct intermission with inspiration. At times, it is altogether absent at the wrists.

Anasarka was occasionally observed in patients, in whom the attack had been prolonged, and who consequently had been sitting for a long time. It was, however, seen only in those, who suffered, at the same time, from varicosities of the lower extremities.

Now and then epistaxis takes place, and greatly alarms the patients.

Physical Signs on the part of the Lungs.

Respiration is always noisy. There is wheezing at the larynx, but rarely tracheal stridor. In some cases, expiration is accompanied by a monotonous and plaintive noise, such as is made in the heaving of heavy weights. In others, the strain is so great, that the patients cry out loudly.

Most of, or all, the auxiliary muscles, which directly or indirectly help towards enlarging or compressing the thorax, are firmly contracted. Their number and force vary, according to the degree of the obstruction. The chest is distended to its utmost limits, always in the antero-posterior, in the transverse and, if the passages are sufficiently pervious, also in the longitudinal diameters. The diaphragm, then, acts forcibly, but normally: descending with inspiration and ascending with expiration.

Frequently, however, the obstruction is so great, that only a limited supply of air is obtained. The lungs are, consequently, unable to follow the muscular traction, but sink in at the apex and at the base. Deep hollows are seen between the projecting sterno-mastoids and scaleni muscles, at the jugular fossa, at the supra-clavicular and at the epigastric regions. For obvious reasons, the diaphragm is now drawn upward on inspiration, but descends on expiration, producing bulging of the abdomen. Its action becomes, thus, of " an inverse type."

So long as the obstruction lasts, there is but slight relaxation of all the muscles. They remain firmly contracted, and hold the thorax in, what is called, the permanent inspiratory position. Inspiration is consequently short, and generally of a peculiar gasping kind. The larynx descends at the same time. Expiration, on the contrary, is prolonged and actively performed, with the aid of all the accessory muscles. But it fails, nevertheless, to depress the ribs and to reduce the diameter of the chest.

As in all obstacles of the air passages, so here, the frequency of respiration is diminished. Its average is between 12 and 20 in the minute; but it greatly varies in different individuals, and in the same patient even during the same attack. In one case were counted, within a space of half an hour, 36, 24, 30, 12, 20; in another 30, 28, 20, 18 respirations per minute; yet no cause was apparent for such changes. An extreme of 56 has only once, that of 7[1] has never been observed. The greater part of the time is devoted to expiration, which occasionally is four times longer than inspiration, so that this form of dyspnœa is of a true expiratory type. Still, in the less severe attacks, the prolongation of the expiration is not so marked.

Vocal fremitus is either completely absent, or of greatly diminished intensity. The difference is especially noticeable in a person with a loud and deep voice.

The results of percussion widely differ, according to the seat and the extent of the obstruction, and to the presence or absence of complications. Uniform increase of resonance, on both sides in front and behind, is rare, and occurs only in the slight attacks. Generally, there is dulness of the upper, and always hyper-resonance of the lower, lobes. The dulness is either on the two sides, or only on one of them; and either in front or behind, or on both. It is most frequently limited to the back—between the supra-spinous fossæ and the angles of the scapulæ. Occasionally, it is absolute and accompanied with increased resistance in the one or the other infra-clavicular region: oftener on the left than on the right.

Stenotic sounds are invariably heard in the air passages. They differ not only as regards their seat and extent, but they are, also, chiefly remarkable for their great mobility. At times, the tidal wave of air is so feeble that no sound whatever is produced within the lungs; and nothing is, then, heard but the laryngeal wheezing, which is conducted downward. In some cases, the chest is full of sonorous and sibilant rhonchi, the latter greatly predominating. In others, there is silence over some parts of the thorax; and over others, there are rhonchi. But in all, these signs rapidly change both their places and characters; silence gives way to rhonchi, and rhonchi to silence in a very striking manner. The sibilant become sonorous, and the sonorous sibilant; either of them may disappear, and vesicular breathing may be heard; and towards the end of the attack, the dry sounds are generally converted into the moist, producing an impression, as if air passed through a gelatinous medium.

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[1] Hyde Salter, *Op. cit.*, p. 77.

In still other cases, as in the following, the stenosis is entirely limited to one side; but, as a rule, it is noticeable only, when the forcible respiratory movements have been subdued by suitable treatment.

Mr. G., æt. 25, a confirmed asthmatic from infancy, was perfectly well on May 4th, 1885. The next morning, at three o'clock, he had one of his attacks. He was seen two hours later, and the following was noted:—Face cyanotic. Respiration noisy, 18 in the minute. Pulse 96, small, firm, paradoxical. Prolonged expiration, inverse type of the action of the diaphragm. Right side, in front and behind, high-pitched percussion note; hyper-resonance on the left. On the right, sibilant and sonorous rhonchi; on the left, loud vesicular breathing, with no trace of an adventitious sound. On the part of the heart, nothing abnormal. After a time the dyspnœa subsided. The upper portions of the right became perfectly pervious, and the rhonchus settled at the base.

Mr. R. B., æt. 12, asthmatic from infancy, was seen in an attack on July 7th, 1886. Respiration 56 in the minute; pulse very small and countless. Diaphragm acted inversely. Hyper-resonance all over. The right side full of sibilant rhonchi; on the left, loud respiration, no rhonchus. In a few minutes, the dyspnœa subsided. The rhonchus entirely disappeared from the right, passing over to the left; but, after a forcible cough, ceased here also.

Notwithstanding the great mobility of the rhonchi, the cough, however forcible it may seem to be, has, so far as can be ascertained, very little influence upon them. Their displacement is, evidently, dependent on the operation of other causes.

SPUTA.

During the paroxysms, while the upper lobes remain obstructed, the sputa, if any there are, consist chiefly of saliva. Expectoration, properly so called, takes place only at the end of them, and varies in quantity and character, according to their intensity and their duration, as also according to the presence or absence of complications.

Thus, four kinds of sputa may be distinguished:—

1. The short attacks generally subside on the removal of a few greyish-white and transparent pellets, from the size of a pea to that of a hazel nut.

2. Where the dyspnœa is associated with catarrhal affections of the larger tubes, there are thick, confluent, exceedingly tenacious masses, of greyish-white colour, slightly frothy, and from two to four ounces in quantity.

3. In the acute forms of the disease, there is a copious, frothy, and perfectly transparent fluid, which contains more or less abundant flocculent particles, of yellowish-white colour, of irregular shape, and of various sizes. Some of them fall to the bottom of the vessel, in which they are collected; others float at different layers of the menstruum, according to the quantity of air, which they contain.

4. In cases of long standing, there are yellow, green or greenish-yellow balls, with the tendency to confluence, and frequently streaked with blood. The quantity may occasionally be very considerable. Hæmoptysis has never been observed during, nor at the end of, an attack. Several patients, however, reported, that they had brought up quantities of blood; but their exaggeration on this point is notorious. Where hæmorrhage was said to have taken place, it was evidently the consequence of certain complications, to which Asthma seems liable; and even then, it occurred only at the intervals between the paroxysms. Hyde Salter states, that a "teaspoonful or so" of pure blood may be coughed up, and that he knew of a profuse hæmorrhage amounting to from half a pint to a pint.[1] It is doubtful, however, whether the case, to which he refers,[2] can properly be called Asthma.

The sputa are inodorous, and show no sign of decomposition. With the exception of the muco-purulent variety, they seem, on the contrary, to be particularly resistant to putrefaction. They may be kept for several days without undergoing any change. Within a week or so, they become liquid, however, and assume a grass-green colour. Rosenbach attributed the latter to the presence of green

[1] *Op. cit.*, p. 89.

[2] *Op. cit.*, p. 369.

spores.[1] From cultivations made of perfectly fresh substance, and with all possible precautions, the colouring-matter appears to be the product of the *Bacillus fluorescens liquefacies* of Fluegge.[2] Troup found that it consisted of various kinds of green and colourless crystals, either agglomerated into drusy masses, or dendritic, ramified, and rigid like leafless twigs; of plates of cholesterine, and of several forms of greenish-brown, yellowish or colourless round bodies.[3]

Their chemical composition varies only as regards the amount of the mucine. In some, this is the preponderating constituent; not so in all. The transparent pellets and the flocculent matter are chiefly hyaline substances; they very slowly dissolve in caustic potash or soda, are not precipitated from this solution by the addition of acetic acid and ferro-cyanide of potassium, and are insoluble in acetic, and imperfectly so, in dilute mineral acids.

If the menstruum is fluid, the form of the solid particles can be readily ascertained, by pouring a small quantity of the sputum on a sheet of black glass, and allowing it to settle. Some of them present themselves, then, as threads of grey or yellowish colour, either straight, or bent, or twisted like a corkscrew; varying in length from one-tenth of an inch to one inch, and in thickness from that of a hair to that of a woollen fibre (Plate I., Fig. 1). By the side of them are small, perfectly transparent gelatinous bodies, like grains of boiled sago, and very tenacious. The larger of them, as also the greyish-white pellets, are covered with layers of mucus, so that it requires some manipulation in order to determine their shapes. The best method of preparation appears to be, to throw them into a 1 per cent. solution of chloride of sodium, and, after a short time, to tease them gently on a glass slide by means of fine camel-hair pencils. In that way, the seemingly shapeless masses may be evolved into solid cylinders; some of them are twisted like a corkscrew, nearly two inches in length (Plate I., Fig. 2); others are straight, knobby, one-eighth of an inch in thickness, and more than three inches in length (Plate I., Fig. 3); others, again, consist of a short, thick stem, an inch and a half in length, with dichotomous division, of which one branch is straight and the other twisted—evidently a cast of a small bronchus (Plate I., Fig. 4); and still others, shorter and thicker than the rest, are so entangled as to resist all efforts to unfold them (Plate I., Fig. 5).

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[1] *Ueber eine neue Art von grassgrünem Sputum: Berl. Klin. Woch.*, No. 48, 1875.

[2] *Die Mikro-organismen*, 1886, p. 289.

[3] *Sputum: Its diagnostic and prognostic Significations.* 1886, p. 84 *seq.*

By the same process, solid cylinders and complete casts may be detected in the catarrhal and muco-purulent sputa, where they are least suspected, although they are, here, comparatively few.

On microscopic examination, these solid bodies are seen to consist of round cells, closely packed together. In the fresh state, there is between the cells a network of delicate fibres, which to some extent disappear with the advance of regressive changes. The casts carry, however, on their surface or within their substance, remarkable threads and cords, which strongly contrast with their surroundings, by their milk-white colour and their strong refraction of light. Some of these are slightly bent (Plate I., Fig. 6, *f*); others are twisted in the most fantastic manner (Plate I., Fig. 6, *a—e*); and all vary considerably, in length, from one-six-hundredth of a line to one line, and, correspondingly, in thickness. Besides, they are evidently much more resistant to decomposition than the cellular elements, in which they are embedded; for when the latter are nearly disintegrated and scarcely recognisable, the former still retain their striking appearance.

The transparent pellets and the sago-like bodies, previously mentioned, are almost entirely composed of those threads. In the catarrhal sputum, these are also found, isolated or rather disengaged, on account of the reduced cohesion of their medium. Most preparations contain a number of them, here also varying in sizes and shapes (Plate III., Figs. 13 A, B, C, and 14; Plate IV., Figs. 20, 21 A, B, 22); some so delicate as to be visible only with a high (Plate IV., Fig. 21, A, B), others so large as to fill up the whole field of a low magnifying power (Plate IV., Fig. 20). In almost all preparations, at least a single specimen will be found. It is necessary only, in order to detect it, to spread the layer, submitted to examination, as thinly as possible—which, owing to the great tenacity of the substance, is often very difficult. In that case, it is desirable to let the prepared slide rest for a while, during which the pressure of the cover-glass effects a uniform distribution. If this means also fails, then the addition of a drop of potash or soda solution will render the thread at once visible; for the mucous corpuscles are soon dissolved, whereas the hyaline substance is only gradually attacked by it.

Equally remarkable is their texture. Some appear to be at first sight perfectly homogeneous (Plate IV., Fig. 21, B), probably owing to their impregnation with mucine; and no trace of structure is, without suitable preparation, discernible in them, even with the highest magnifying power. Generally, however, they plainly show that they are made up of strands and fibres. They are woven upon the plan, if one may say so, of the figure of 8. The closeness or looseness of their texture accounts for their varying appearances. Some retain their cohesion, but have fibres projecting along their surfaces, so that they look like cords of some coarse material (Plate III., Fig. 13, A, B, and C). As a rule, they readily unwind. The coil gives way at some parts (Plate IV., Fig. 21, A), and the finer specimens may occasionally be seen entirely untwisted (Plate III., Fig. 15). If their ends happen to overlap, they then appear somewhat like a St. Catharine's wheel (Plate V., Fig. 25). In the larger and thicker cords, however, this change either takes place uniformly throughout (Plate IV., Fig. 22); or it commences at the periphery, and proceeds towards the centre (Plate II., Figs. 7 and 8). The outer layers thus become loose (Plate II., Fig. 7, *b*, *c*), and frill out at the end (Plate II., Fig. 7, *d*), whereas the core (Plate II., Figs. 7 and 8 *a*), as it were, continues to be tightly wound, and is as yet not implicated in the process.

In that state, they represent the central thread and outer spiral, described by Curschmann.[1] At last, however, the whole structure is unfolded (Plate IV., Fig. 22); its fibres break up into fragments, and their interstices are invaded by small round cells. Heaps of such cells are seen to force their way into the meshes of the cords (Plate II., Fig. 7, *b* and *c*), apparently hastening their disintegration.

As regards their chemical composition, it has already been mentioned, that they are but slowly dissolved by caustic potash or soda. Acetic acid frequently makes them more transparent, and causes them to swell; often it has, even in strong solution, no effect whatever upon them. With Lugol's solution, they become faintly yellow; with picro-carmine, they take the picric acid; they stain readily with eosine and saffranine, but obtain a reddish tint from gentian-violet, and a dirty violet from hæmatoxyline.

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[1] *Deutsch. Arch. f. Klin. Mediz.*, vol. xxxii.

The fibres, which make up the more delicate threads, are generally of uniform thickness, strongly refracting light, disposed to break up into small rods (Plate III., Fig. 15), and some of them provided with a roundish enlargement at one end (Plate IV., Fig. 21, A). An additional and different set enters into the formation of the thicker cords; some of them are very fine and longitudinally arranged (Plate V., Fig. 26^a); others are of varying thickness, more or less irregularly spiral, and generally transversely placed (Plate V., Figs. 24^c and 26^a *e*).

The meshes between those fibres (Plate V., Fig. 26^a) contain:—

1. Leucocytes in different stages of disintegration; their protoplasm is apparently altered in its composition: those that approximately retain their original form are either elongated or spindle-shaped; the greater part of them have apparently run together, forming into irregular masses; they stain but faintly with aniline dyes, and are filled with a streptococcus (Plate III., Fig. 16^b).

2. Charcot's crystals, in varying numbers and sizes, lie either longitudinally or transversely, singly or in heaps (Plate IV., Fig. 23).

3. Chains of a streptococcus, of which each individual

measures about 0·4 μ, and stains after the method of Gram (Plate III., Fig. 15^a; Plate V., Fig. 26^a *d*; Plate VI., Fig. 27).

4. A small bacillus, with rounded-off poles, slightly knobby, between 2—4 μ in length, and about 1 μ broad, staining also, with the exception of one spot in the centre, after the method of Gram, and arranged in loops (Plate V., Figs. 26 and 26^a *e*). With a higher magnifying power (Zeiss $\frac{1}{12}$ Im., Oc. 18) some individuals appear to be oval, faintly stained in the body, but deeply so at its poles;—while others are round, much smaller, and apparently altered by involution (Plate VI., Fig. 28). Both, the bacillus and the streptococcus, were found in perfectly fresh sputum, which had been collected in sterilised tubes, after the mouths of the patients had been, as far as possible, disinfected by solutions of borax and corrosive sublimate.

With the advance of disintegration, when, as already mentioned, the cords and threads begin to uncoil, the contents of the meshes pass from them into the more fluid medium. The micro-organisms above described are then the first to disappear, and their place is taken by various putrefactive bacteria. In cover-glass preparations, made at the time, the streptococcus will rarely be found, whereas the bacillus may much longer be detected. Charcot's crystals, however, being much more resistant, will be seen after their release, as it were, more readily, and in greater abundance than before; and this circumstance accounts for the erroneous supposition of Ungar, that they are developed when the sputum is kept in a moist chamber for about three days.[1]

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[1] *Verhandl. d. Congress. f. Inn. Med.* 1882.

Some of the threads, visible with the naked eye (Plate I., Fig. 1) are seen, on microscopic examination, to be spirally twisted and coarsely striated (Plate II., Fig. 10). When unfolded, they appear to be shreds of pseudo-membrane, which are derived from the alveolar portion of the lungs. They present casts of the infundibula and of the air-vesicles (Plate II., Fig. 11), and contain concentric layers of micro-organisms, which resemble the micrococcus of pneumonia (Plate III., Fig. 12). The micrococcus has a distinct capsule, and is arranged either as a diplococcus or in short chains. Chemical analysis, and their reaction to stains, show the shreds to be composed of hyaline substances.

Fibrinous and hyaline fibres made up into bundles with radiating branches (Plate V., Figs. 24, 24^{a}) are frequently met with in the catarrhal sputum. With a high magnifying power, they are seen to consist of a network of fibres, varying in length and thickness; some of them extremely delicate, others breaking up into small rods and still smaller globules. The meshes, here also, contain a streptococcus (Plate V., Fig. 24^{b}).

In the more fluid kind of the sputa, there are generally large masses of cylindrical epithelium. The fact, mentioned by Lewy,[1] that their number is in an inverse ratio to that of Charcot's crystals, is probably correct; but the interpretation of it is evident from what has previously been stated. The epithelium is frequently so closely packed together as to form casts of bronchi, with their dichotomous division (Plate II., Fig. 9), which carry on their surface bundles of fibrine threads (*a*). Often, spindle-shaped

[1] B. Lewy, *Ueber d. Beziehung. d. sogen. Spiralfäden u. Asthma Kryst. z. Asthma: Zeitsch. f. Klin. Mediz.*, ix. Bd., 6 Heft.

epithelium in various stages of degeneration surrounds the hyaline cords. Some of those cells are isolated, and greatly altered in their appearance and chemical composition (Plate III., Fig. 16). As a rule, they undergo peculiar changes; the pointed end begins to lengthen, and they are gradually drawn out into thin fibres at the expense of their bodies (Plate III., Fig. 16^a, Plate IV., Fig. 17), which at last entirely disappear. Alveolar epithelium is of comparatively rare occurrence. If so, it looks in cover-glass preparation as though worm-eaten, the holes having been apparently occupied by various forms of micro-organisms, which have subsequently fallen out.

In the muco-purulent variety of the sputum, the peculiarities, hitherto described, are but faintly marked, possibly owing to the solvent influence of the menstruum. Neither the micro-organisms above mentioned,[1] nor the crystals, can be found in it; only the cords resist, to a certain extent, disintegration, so that fragments of them, either still twisted or unwound, are almost always visible. If uncoiled, they appear as fibres arranged in a longitudinal series, with others lying crosswise upon them.

In some cases (5 out of 180) tubercle bacilli were detected in the catarrhal sputum.

SYMPTOMS OF THE INTERVALS.

The expectoration of the characteristic sputa always brings some relief, and with it comes the welcome feeling, that the air-passages are again pervious. Respiration becomes freer, less noisy, and quicker; the distension of the chest gradually yields. The vesicular breathing is again audible, where complete silence previously existed. At some places, air appears passing through a gelatinous or liquid medium; the rhonchi diminish in number and sound less dry. At last, they seem to settle chiefly toward the base. There is a great deal of exhaustion, with inclination to sleep.

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[1] *Vide* p. 58 *seq.*

In the acute form of the disease, the improvement is only imperfect. Within a few hours, the dyspnœa generally returns with its former intensity. Such a series of severe paroxysms continues, with but slight intermissions, till the inflammatory process has subsided, and till the inflammatory products have been removed.

In the chronic form, recovery may be, in some cases, complete. Not a trace of a morbid sign can be detected, and the impression obtrudes itself, that the attack, although exceedingly painful, was after all a depurating and salutary effort. The patient looks pinched, from the copious loss of water by perspiration and salivation; his muscles feel bruised, from the long strain upon them; the tongue is coated, the appetite is bad, and there is a feeling of flatulence. All these symptoms, however, entirely subside within a day or so, and the previous suffering is soon forgotten.

In other cases, however, the subjective freedom of respiration is, indeed, considerable; but the physical signs show the persistence of the pathological process. There is slight cyanosis; the respiration is shallow, and the diaphragm continues, for days, its inverse type of action. The cough is occasionally troublesome; and the sputa, copious at first, become scanty, but retain their peculiar characteristics. Expectoration hardly ever ceases, although it may be limited to a few pellets, which are raised in the morning.

Over a greater or lesser part of the thorax, the various signs of stenosis persist. But their mutability is such as to render it impossible to make a general statement on their comparative frequency, at the one or the other spot. An idea of their disposition to change may be obtained from the following case.

Mrs. H., æt. 49, had just recovered from a severe attack of Asthma, and her breathing was subjectively quite free. She presented, however, the following signs:—

August 1st, 1880.—Right infraclavicular region, resonance good, sub-crepitant rhonchus. Right base in front and behind, resonance good, distant rhonchus, no vesicular breathing. Left perfectly normal.

August 2nd, 1880.—The right side, in front and behind, dull resonance and complete silence. Left side, normal resonance and loud vesicular breathing.

August 3rd, 1880.—The right side is completely cleared up. On the left, resonance is impaired, and respiration very feeble.

In some cases, there is persistent sub-febrile temperature. The patients, consequently, dislike warm rooms, and sit, even in the winter, without a fire, alleging that the heat produces an attack.

DIGESTION.

With the cessation of the dyspnœa, the appetite, as a rule, returns. In advanced cases, however, with a great deal of mischief in the lungs, with persistent cyanosis and obstruction of the bronchi, the patients are afraid to eat, for fear of bringing on an attack. There is, indeed, after a heavy meal, some difficulty in breathing; for, owing to the increase of the respiratory wants after food, the respiratory movements become deeper and more frequent in order to supply them; and, while at rest and with superficial respiration everything went well, the effort is now painfully perceived.

Constipation is the rule. Diarrhœa is rare. Generally, there is much discomfort in the intestinal canal—discomfort, due partly to products of faulty digestion, partly to the persistent wind-sucking.

The tendency to nausea lasts, in some cases, for a long time, and is accompanied by a feeling of faintness. It probably depends upon pharyngitis, and seems to arise from a firmly adherent layer of mucus.

DURATION OF THE PAROXYSMS.

In an affection like the present, where the suffering is so terrible, and the symptomatic treatment, upon the whole, so effective, it is difficult to form, from actual observation, a definite opinion on the probable duration of the paroxysm. According to the reports of the patients, the attacks may last from a few minutes to several days. One of them declared, that he had had, repeatedly, to stand for two successive days and nights, panting for breath; another, that he had spent several weeks in his armchair, suffering all the while, with but slight interruptions, from the greatest dyspnœa. There can be little doubt on the accuracy of the statements, although some allowance must be made for the elasticity in the diagnosis of Asthma. It is not improbable, however, that those statements apply to the series of paroxyms, such as compose an access of the acute form of the disease; for, unless the inflammatory process be completely subdued and the obstruction be entirely removed, the dyspnœa may return within a few hours, during which the patient has barely time to recover. As a rule, an acute attack, with its ups and downs, continues rarely longer than a week, provided only, that the patient can be sufficiently controlled.

TIME AND MODE OF ACCESS.

The dyspnœa is apt to occur at all hours of the day and the night; perhaps with greater frequency during the latter; and here, again, as often before as after midnight. Whether and what influence sleep as such has on the production of it, would be mere matter of speculation. Any theory based upon the observation, that drowsiness often precedes the outbreak of an attack, is untenable for this reason—that the one and the other are co-ordinate results of the same cause. In some instances the origin is quite sudden; in others, however, the attack gradually develops itself, and reaches, from within a quarter of an hour to several hours, its maximum degree of intensity.

FREQUENCY.

Some patients allege, that their dyspnœa returns every hour; but they are generally those who, at the least sign of irritation in the throat, fly to their pet remedies, and describe the slightest feeling of constriction at the chest as a paroxysm of Asthma. In the chronic form of the disease, it is said, that not a day passes without an attack; if so, there is no such inherent disposition in it, but that frequency may be favourably modified by suitable treatment.

In the acute form, there is always, as already mentioned, a series of attacks. The obstruction, if once removed, is

ALLEGED ALTERNATIONS WITH OTHER DISEASES.

It is commonly held that Asthma is favourably influenced by the accession of other pathological conditions. Thus, *e.g.*, cases are reported, in which the dyspnœa is said to have been relieved by gangrene of the intestines, and by a fracture of the ribs in consequence of a fall from a horse. As a rule, that theory rests on statements by patients, who, in matters of that kind, are not reliable guides even in respect of facts. One declared, that his skin was always more irritable in the intervals between his paroxysms; and when, during them, he was seen rubbing his face, he maintained that the itching was always more intense at that time. Another, who had a meningocele in the left lambdoidal suture, stated, that it entirely subsided during the attacks; yet no change in its size nor in its shape was perceptible when there was occasion to witness them.

Trustworthy observers, however, maintain that exacerbations and remissions of the dyspnœa frequently coincide with the appearance and cessation of epilepsy, gout, rheumatism, and of skin diseases. A sudden cessation of an asthmatic paroxysm is, under all circumstances, quite intelligible, from what is known of its nature. Moreover, the distribution of the blood is so greatly disturbed during a seizure, that an existing affection may conceivably be benefited by it. As regards skin diseases, it is true that an eczematous eruption which has nearly faded, reappears with the return and persists after the cessation of the Asthma. But the question is, whether Asthma is relieved by the skin disease, or whether the skin disease is aggravated by Asthma. There is no evidence that, upon the whole, such occurrences are more than isolated facts, and that there are, in any given case, distinct and constant alternations. Nor is there proof that two such events are necessarily connected, and that both proceed from a common cause.

CHAPTER III.

PATHOLOGICAL ANATOMY OF BRONCHIAL ASTHMA.

Results of Post-mortem Examinations—Essential Changes of the Respiratory Surface.

RESULTS OF POST-MORTEM EXAMINATIONS.

ON account of the protracted but fitful course of their disease, asthmatics are, rarely, inmates of hospitals; and as post-mortem examinations are only exceptionally performed in private practice, the anatomical data on the subject are, consequently, very scanty. The results of the autopsies that had been made and published up to within the last thirty years, have been collected by Thery[1]; but if they have any value, they merely show that a form of paroxysmal dyspnœa may occur in connection with all conceivable diseases. Since that time, only five such examinations have been reported:

In the case of an old man, aged 76, who had suffered from chronic bronchitis with Asthma,—embolism of the pulmonary artery was found.[2]

A man, aged 59, had been, for more than 30 years, subject to hay fever with chest symptoms, and died suddenly on leaving a very hot sulphur bath. The lungs were distended, the trachea and bronchi filled with a frothy fluid, the mucous membrane was red and swollen, and the heart was hypertrophied and covered with fat.[1]

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[1] *Op. cit.*, p. 199.

[2] Virchow, *Gesammelte Abhandlungen z. wissensch. Mediz.*, 1856, p. 356.

A strong healthy labourer, aged 36 years, presented symptoms of tetanus—loss of consciousness, distortion of the face, opisthotonos and clonic contractions of the muscles of the limbs—symptoms which were ushered in by a kind of epileptic cry, and were accompanied by intense dyspnœa. He died in one of those attacks. The lungs were congested, and the heart was hypertrophied and dilated.[2]

In a young man, asthmatic for many years, there was extensive bullous emphysema,[3] a portion of the upper lobe of one side having developed into a large sac containing air.

All these cases, however, are, for various reasons, inconclusive, and afford no information on the pathology of the one particular form of asthmatic paroxysms.

Of greater importance is the one which recently occurred in Leyden's practice.[4]

A lady, aged 40 years, had, from earliest infancy, been subject to typical attacks of Asthma. Her constitution had been undermined by care and anxiety. She was, also, in the highest degree, addicted to the abuse of morphia. The disease in her case assumed, of late, an unusual intensity, and was complicated by dropsy and albuminuria, which indicated the near approach of the end. The lungs were emphysematous. The respiration was feeble, and sibilant rhonchi were heard all over the chest. She had, daily, several paroxyms of dyspnœa, which presented the characteristic signs, and only differed in number and severity. The sputum was that generally observed in fresh cases. Its quantity varied. It was very viscid, of a greyish-white colour, spumous, and contained, almost always, long, whitish, fibrinous threads, but very few greenish and globular masses. Death took place during one of those prolonged attacks, with cyanosis and stertor.

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[1] Phœbus, *Der typische Fruehsommer-Katarrh*, 1862, p. 69 *seq.*

[2] Bamberger, *loc. cit.*, p. 102.

[3] Fraentzel, *loc. cit.*

[4] E. Leyden, *Ueber Bronchialasthma. Vortrag gehalten in d. militär-ärztlich. Gesellschaft.* Berlin, 1886, p. 13, *seq.*

The autopsy, performed six hours after death, showed extensive vesicular emphysema. Both lungs were very large, over-distended, pale, and their margins were partially covered with large bullæ. The bronchi were not dilated, their mucous membrane was red, but not materially altered. The bronchioles contained small quantities of mucus; and greenish or whitish substances, in which neither fibrine nor crystals were found, could be squeezed from the cut surface of the lungs.

Sections were made of the organ after it had been hardened in alcohol. Amidst a number of dilated alveoli, there were several which were normal. They were filled with granular (grumous) masses, which contained a moderate amount of large cells. The walls of the bronchioles were not materially altered; but layers of a coarsely granular, apparently amorphous substance, which considerably reduced their calibres, firmly adhered to the surfaces of their mucous membranes. In one bronchiole, the exudation was annular, with only a narrow slit in the centre; in another, it had somewhat the shape of a corkscrew, leaving a semilunar passage.

In the 180 cases, which were under my own observation, there was once only the opportunity of making a post-mortem examination.

Mrs. C., æt. 37, with a history of phthisis on her father's side, had had, when a child, whooping-cough, measles, and scarlet fever. She had had, also, as she distinctly remembered, a marked disposition to colds, which were especially severe in the early summer. Menstruation began at the age of 13. She married at the age of 21, and has had five children, all of whom are alive. Fourteen years ago, she had been seized with an attack of bronchitis, in consequence of which she had been confined to bed for several weeks. Recovery had been but imperfect; her breathing had remained embarrassed, and soon afterwards she had become subject to typical attacks of Asthma. The bronchitis had frequently returned, lasting each time for several weeks, during which she had been unable to leave her bed. Early in June 1886, her legs had begun to swell, and in September of the same year she sought advice at, and was admitted into, one of the Metropolitan hospitals.

From the notes which the Registrar of the Hospital kindly permitted me to peruse, it appeared that, on admission, the urine was diminished in quantity and contained albumen. The heart's action was disturbed; stenotic sounds were heard all over the chest, and she had severe attacks of Asthma, which were, but slightly, relieved by fumes of burning nitre paper, and by stimulants.

She left the hospital on the 6th of January, 1887. The œdema was considerably reduced, but the dyspnœal paroxysms persisted in all their intensity and frequency.

She was seen on January 14th, 1887, when she complained of: dyspnœa, cough, insomnia, diarrhœa, and weakness. The following was noted: Small stature, fairly nourished, rickety shape of head, œdema and cyanosis of the face, and anasarka of the legs. Thorax narrow and flat; lordosis of spine. Respiration 20 in the minute; inverse action of the diaphragm. Resonance, both sides, short and high-pitched. Sonorous and sibilant rhonchi all over. Pulse 100, small, soft, and regular. Epigastric pulsation. Impulse distinct in fifth interspace inside nipple line. Cardiac dulness commencing at fourth rib above, and transversely extending to beyond right sternal border. Sounds loud and clear. Tongue cyanotic, clean. Abdomen distended. Hepatic dulness could not be made out. No appreciable enlargement of the spleen. Urine of low specific gravity, no albumen. Sputa about 4 oz. in 24 hours, of greyish-white colour, very viscid and frothy, containing all the elements, previously described.

She was, again, seen Jan. 23rd, 1887. Her nights had been somewhat better, and the cough and the breathing had also slightly improved. She had taken some food, and had endeavoured to attend, as far as she was able, to her domestic duties. But for the last two days, all the symptoms had grown worse; she had also passed less water, and the swelling of the legs had increased.

The cyanosis was very considerable. Her hands were cold, and there was no pulse at the wrists. The dyspnœa was very great. Resonance on both sides was wooden, and there was complete silence on the right, but rhonchi were heard on the left. The cardiac impulse was neither seen nor felt, and the cardiac sounds were very feeble.

Powerful stimulants had no effect, and she died the next day at 1 o'clock a.m.

Post-mortem[1] (twenty-four hours after death) :--

Considerable hypertrophy and dilatation of the heart. Valves intact; aorta very narrow, containing patches of atheroma. Lungs not collapsed, dark brown, rather dry; extensive adhesions of the right apex; collapse induration of the right base. No signs of inflammation.

In the right main bronchus was found a spongy-looking cylindre, of a dark brown colour, and of a dry and somewhat friable material (Plate VI., Fig. 29). It lay loosely within the tube, and from its upper third was given off a fine branch, which was prolonged into a bronchiole (Plate VI., Fig. 29 *a*). It nearly filled up the tube, so that but little air could have passed between it and the bronchial wall. The left main bronchus contained a small quantity of greenish, soft mucus, and in the one of its divisions, there was also a cylindrical formation like the one just described. The smaller bronchi were apparently dilated; their surfaces were dark; and a great many of them were, partially, occluded by exudation. Both lungs were emphysematous.

There was some ascites. The liver, spleen and kidneys were large, pale and dry, and showed signs of amyloid degeneration.

The right main bronchus with its contents and portions of the lungs were hardened in absolute alcohol, and then submitted to microscopic examination.

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[1] The post-mortem was performed in a small, dark room, in one of the alleys leading out of Berwick Street, Oxford Street, W. Mr. Charles J. B. Carter, M.R.C.S., etc., and Mr. Edwin H. Light, M.B., etc., Camb., were good enough to assist me.

The brown cylindrical mass consisted, chiefly, of cylindrical epithelium, in various stages of degeneration, and greatly deformed (Plate III., Fig. 16, and Plate IV., Fig. 17). Here and there were found the milky-white cords, previously mentioned (Plate III., Fig. 13 A, B, C, and Plate IV., Fig. 21 A, B). Charcot's crystals were not seen.

On sections of the right main bronchus, polypous excrescences were seen, projecting from the mucous membrane (Plate VI., Fig. 30). The growth consisted of a firm fibrous tissue, and contained no vessels; its free end was exfoliated (Fig. 30 *b*), and its sides were coated with a single layer of a mis-shapen, oblong epithelium. The surface of the mucous membrane was almost entirely denuded of epithelium: only a few spindle-shaped cells were seen standing here and there. The basement membrane was broad and of a hyaline aspect. Between the folds of the internal fibrous layer were colonies of staphylococcus. A deep ulceration (Plate VI., Fig. 30 *a*), extended to nearly the surface of the cartilage. There were no signs whatever, that the loss of substance was of a tubercular origin. The vessels (Plate VI., Fig. 30 *d*) were rather large. The mucous glands in the wall of the bronchus were, remarkably, few in number, and very small (Plate VI., Fig. 30 *c*, *c*), looking as if they were atrophied.

The smallest bronchi were dilated and entirely denuded of epithelium. Some contained fibrinous threads stretched across their walls (Plate VI., Fig. 32 *a*); and between those threads were colonies of streptococcus. Others had an ampulliform dilatation at one end (Plate VII., Fig. 33 *a*), and a peculiar S-shaped deformity at the other (Fig. 33 *c*).

The rest of their walls was apparently not much altered. They contained masses of exudation (Plate VII., Fig. 33, *a*, *b*, *c*), which was, almost entirely, made up of colonies of streptococcus (Plate VII., Fig. 34).

The bronchi, of medium size, showed, both in transverse (Plate VI., Fig. 31), and in oblique sections (Plate VII., Fig. 35), irregular dilatations. Their walls were slightly thickened. In some (Plate VI., Fig. 31) there was a considerable hyperplasia of the mucous membrane. Most of them were more or less occluded by masses of detritus, in the centre of which no micro-organisms could be detected; but there were fragments of Charcot's crystals.

The alveoli were emphysematous. They contained no trace of epithelium anywhere, but some of them were nearly filled with a fibrinous exudation. Their walls had many dehiscences, where, at some places, the inter-capillary nuclei still lay loosely; and there was a great deal of pigment. The capillaries were dilated, and naturally injected; but no micro-organisms could be detected in either the blood or the lymphatic vessels.

Thus, the pathological changes, which the post-mortem revealed in that case, were:—dilatation of the smaller bronchi, with collapse induration at some parts; emphysema of the alveoli; diffuse exfoliation of the epithelium, and fibrinous exudation within some of the bronchioles and alveoli; and partial or complete obstruction of both small and large tubes by masses of detritus.

To conclude from this one instance, as to the probable state of the lungs, in all cases of Asthma, would, indeed, be inadmissible; yet its importance will be fully appreciated, on due consideration of the fact, that the clinical phenomena of the chronic forms of the disease could not, possibly, arise in circumstances, materially different from those here described.

ESSENTIAL CHANGES OF THE RESPIRATORY SURFACE.

A tolerably accurate idea of what constitutes the essential feature of the pathological process may be obtained by the study of the pathological products. So far as an inference from them is possible, it appears that the epithelium is the starting point of the morbid action. It exfoliates from the greater part of the respiratory tract, especially the larger bronchi, and is then, as already mentioned, seen in the sputa, not only altered in shape, but often in coherent membranes, so as to represent the form of the tubes (Plate II., Fig. 9).

In the smaller bronchi, it undergoes peculiar changes: it is gradually drawn out into fibres, like a piece of metal is into wire; thus altered, it betrays its origin merely by a slight enlargement at one end (Plate IV., Fig. 19). This elongation is, probably, effected by the drooping of the heavier body of the cell, which drags upon its inserted portion. In that way the fibres on opposite sides of the walls meet, interlace and form some of the threads previously described (Plate III., Fig. 16*a*).

The desquamation of the epithelium is, as usual, followed by a copious emigration of leucocytes, which, as shown by the expectorated casts (Plate I., Figs. 3—5), may be so considerable as to completely occlude tubes not exceeding one-eighth of an inch in diameter. In contact with the fibrino-plastic substances, which exude at the same time, the white corpuscles disintegrate. Most of them are converted into fibrinous and hyaline threads (Plate V., Fig. 24, *a, b, c*), while the meshes which they form inclose those cells that resisted the dissolution (Plate V., Fig. 26*a*). Together with the degenerated epithelium, they compose the larger kind of the cords (Plate V., Fig. 26*a*).

The subsequent metamorphoses of the leucocytes are determined by the anatomical conditions of the surface upon which the exudation takes place. In the alveoli and in the bronchioles, they retain their hyaline character, and enter into the structure of the pseudo-membranes (Plate II., Figs. 10 and 11), and of the cords. In the larger bronchi, however, they are subject to the solvent influence of the secretion of the mucous glands, so that, the nearer the centre, the more their origin is obliterated.

The softened exudation may be expectorated, if the "*vis a tergo*" is sufficient for the purpose. But if, on account of its tenacity, it is retained within the air passages, it becomes the nidus of certain micro-organisms which, by their specific action, continue the softening and convert it into greenish and less viscid masses. Some of the hyaline portions, however, are more than others resistent to that peptonising influence, and, thus, they are still found in the sputum.

As a rule, the threads, which develop in the bronchioles, are readily expelled, when once detached from their matrix, provided that the passages above them are pervious. Otherwise, they are arrested in their onward movement; they are either invaginated, or are seen lying upon or within the contents of the larger tubes (Plate I., Fig. 6).

Nothing can be definitely ascertained as to the extent of the fibrinous exudation,—whether it be limited to a few lobules, or whether it extend over one or several lobes. From the size of the casts, hitherto observed, it apparently takes place in tubes not exceeding one-eighth of an inch in diameter. From here the process descends to the periphery, involving, occasionally, also the alveolar surface. Probably the larger bronchi are equally affected, but in a less degree; if so, the exudation is readily dissolved and then loses its pathognomonic aspect; or it is soon expectorated by cough, so that no time is allowed for coagulation.

CHAPTER IV.

PATHOLOGY OF BRONCHIAL ASTHMA.

Nature of the Disease—Nature of the Dyspnœal Paroxysms—Their Mode of Cessation—Terminology.

NATURE OF THE DISEASE.

THE sputa in Asthma afford ample evidence, by their structure and their chemical composition, that they are the product of a fibrinous exudation into the smaller bronchi. And their invariable occurrence at the end of the dyspnœal paroxysms plainly indicates, that they are in some way connected with the disturbance of the respiration. Only in one form of the disease—the so-called primary or essential—are they said to be absent, the mucous membrane being, presumably, quite free from even the slightest organic lesions.[1] But that form has been established without any clinical proof; and it has, confessedly, been accepted out of deference merely to traditional views.[2]

Actual observation, however, gives no support to that theory. It would be difficult to demonstrate a case in which the dyspnœa presented all the postulated characteristics—such as distension of the chest, diminished frequency of respiration, tympanitic resonance, prolonged expiration, and sibilant rhonchi—without being accompanied by the pathognomic sputum. Till the contrary, therefore, has been conclusively shown, the constant association of that peculiar pathological process with the functional derangement must be maintained as an indisputable fact.

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[1] Riegel, *Loc. cit.*

[2] Curschmann, *Verhandl. d. Congress. für Inn. Mediz.*, 1885, p. 245.

The question now arises as to the probable nature of their connection. It would seem superfluous to insist, at the outset, that the exudation was always present before the appearance of the attacks, had it not been suggested that it was a consequence of them, and due to a vaso-motor neurosis. The sequence of the phenomena is readily traceable in practice; but even, if there is no opportunity of examining a patient, the hypothesis is obviously untenable. Granted that a croupous exudation may arise from the alleged nervous disturbance—a supposition as yet devoid of all foundation—it could not possibly have such an origin in the present circumstances. For, from what is known of the visible mucous membranes, the product of a catarrhal or croupous inflammation requires at least several hours to form. Therefore, if an attack spontaneously or by treatment subsides in less time, the expectoration must have developed during, or must have existed previously to it. The same still more forcibly applies to cases in which the sputum has already undergone regressive metamorphosis.

But the answer would be incomplete, if it merely referred to the dyspnœa and the exudation. For, though the dyspnœal paroxysms are the more conspicuous, they are not the sole, manifestation of the disease. If, in the chronic forms, they seemingly constitute it by themselves, their development shows, that they are only an episode, so to say. In the acute and typical forms, there is yet another series of symptoms by which they are invariably preceded. They are ushered in—as will be remembered from the description of the clinical history—by constitutional disturbances, by severe irritation of the upper part of the respiratory tract, and not unfrequently also by cutaneous eruptions. Consequently, there must be some relation between the one group and the other.

According to the prevalent theories, those prodromal symptoms are supposed to be idiopathic affections—colds, hay fever, and vaso-motor disturbances—which merely act as exciting causes. Their clinical peculiarities, however, are inconsistent with the current views of their natures. They are, evidently, the result of an inflammatory process, which commences in the pharynx, travels upwards to the nose and to the eyes, and downwards into the air passages. Now, neither mechanical irritations nor a derangement of innervation would spread from place to place. The effects, which they produce, would be limited to their points of impact.

It follows, therefore, that those symptoms cannot possibly arise in the manner generally alleged. Besides, as previously mentioned, croupous membranes are, in those cases, often observed on the pharynx, on the tonsils, and especially on the tongue, the epithelium of which exhibits, at the earlier stages of the disease, a marked fibrinoid degeneration.

Thus, the pathological process on the upper portion of the respiratory organs appears to be identical with that on its lower portion. The conclusion, therefore, seems irresistible that, what is commonly described as Bronchial Asthma, is an acute and progressive, nay, almost erysipelatous, form of inflammation, which extends from the pharynx upwards and downwards, and is accompanied by a croupous exudation.

When the disease has once declared itself, it becomes habitual, resembling, in that respect also, a certain form of erysipelas. The tendency to recurrence is, no doubt, mainly due to the persistence of a peculiar predisposition; but, in some measure, it is increased also by the attacks themselves, which enhance the vulnerability of the tissues. The surface of the mucous membrane, gradually, loses its power of reaction, by means of which it rids itself of the special morbific agent. The signs of irritation, which are, at first, the more obvious, become in the course of time less acute, whereas those of exudation assume a greater prominence.

NATURE OF THE PAROXYSMS.

The dyspnœal paroxysms are, thus, a symptom merely of a peculiar inflammatory affection. That the croupous exudation is concerned in their production, can hardly be doubted. However variously the origin of the physical signs may have been interpreted, this much has been generally admitted—that they point, in an unmistakable manner, to a stenosis of the air passages.

The process, it has rightly been urged, is to all appearances not sufficiently extensive to cause serious obstruction of the smaller air tubes. To do so, it would be necessary that it should be at least as diffuse as capillary bronchitis. But, on the cessation of even the most intense dyspnœa, the quantity of the sputa is often found to be very small. Therefore, it could have been limited only to a few bronchioles; and as it apparently affects only those, the diameter of which does not exceed an eighth of an inch, it is impossible to assume, that their occlusion could produce the dyspnœa. A reduction of the respiratory process to so slight an extent would readily be compensated by the tubes, which remain pervious. Nor, indeed, would it account for the fitful appearance of the attacks, and their sudden onset and subsidence.

The theory, advanced by Curschmann, that the sputa cause irritation of the terminal fibres of the vagus, and thus a bronchial contraction is, apart from the impossibility of the alleged spasm, disproved by facts. The sputa, as well as the stenotic signs, persist during the intervals, in which respiration is almost or entirely unimpeded.

The occasional preponderance of sibilant rhonchi has given rise to the generally received opinion, that the stenosis resides within the smaller air tubes. Although those sounds are, unquestionably, produced at narrow orifices, there is nothing to warrant the assumption, that the orifices must be in the small, but may not be in the larger bronchi. On the contrary, all the physical signs, taken together—the distension of the chest, the hyper-resonance on percussion, the sinking in of the supra-clavicular and of the epigastric regions on inspiration, and the absence of vocal vibration—all these point to the centre, as the seat of the obstruction.

There can be little doubt, that the immediate cause of the dyspnœal paroxysms is a transient stenosis of the main bronchi, or even of the trachea. Its mode of production resembles, in many respects, thrombosis and embolism. As in the bloodvessels, so here, the function is, in no way, disturbed so long as only a limited number of tubes is obstructed. The exudation is harmless, so to say, at its place of origin; and its presence does not even betray itself by cough, which is not induced by irritation of the surface of the bronchioles. Gradually, however, the exudation shrinks and undergoes other regressive changes, in consequence of which it becomes detached from its matrix and is rendered movable. Air can now pass between it and the bronchial wall. The *vis a tergo* thus created, although sufficient to displace it, is too slight to effect its expulsion. Owing to its great tenacity, the plug is, in its onward course, arrested, and however small it may be in size, it necessarily diminishes the calibre of even a large bronchus. Normally, the diameters of the trachea at its bifurcation vary: in the frontal direction between 0·96 and 0·70 of an inch, and in the sagittal between 0·96 and 0·68 of an inch; those of the right main bronchus: between 0·81 and 0·48 of an inch; and those of the left: between 0·68 and 0·36 of an inch.[1] In the asthmatics, however, there are grounds for supposing that those measurements are rarely attained;[2] and it is evident, therefore, that a considerable stenosis may thus be readily produced.

The larger the obstructed tube, the more difficult and imperfect is, for obvious reasons, the necessary compensation. All the symptoms of the asthmatic paroxysm become thus intelligible. The subjective dyspnœa and the forcible respiratory movements are chiefly the consequence of the deficient ventilation of the lungs; and the diminished frequency of respiration results from the obstacle within the air passages. Both inspiration and expiration are impeded. But the plug, coming as it does from below upwards, acts in the manner of an expiratory valve, allowing the access, but preventing the egress, of air. The thorax is, as far as possible, distended to its utmost limits; but by the imperfect relaxation of the inspiratory muscles, expiration is deprived of its main source of energy, so that, though prolonged and actively performed, it is, nevertheless, ineffectual. This modification of the respiratory movements, is due to the increase of the carbonic acid, and to the diminution of the oxygen, in the blood, which, thus altered, powerfully excites the centre.

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[1] Aeby, *Der Bronchialbaum d. Säugethiere u.d. Menschen*, 1880, p. 67 *seq*.

[2] Benecke, *Die anatom. Grundlagen d. Constitutions Anomalien*, 1878.

To some extent, those forcible respiratory movements proceed also from the irritation of the sensory fibres of the vagus. As in all hollow organs—for instance, the rectum and the bladder—so in the air-passages, the presence of a foreign body at or near their orifices causes unsuitable contractions of the muscles which regulate their capacities. The embolic plug, arrested in one of the main tubes, acts in the manner here suggested, and produces what may be termed a "bronchial tenesmus."

MODE OF CESSATION OF THE PAROXYSMS.

Although the inspiratory traction fails in supplying the requisite quantity of air, it has, nevertheless, a certain salutary effect. By enlarging the thorax, it rarefies the stagnant air behind the obstacle, and thus leads to an increased exudation of serum from the dilated vessels in the bronchial mucous membrane. There is, consequently, a *hydrops ex vacuo*, as it were, which, by the flushing of the tube, serves to remove the obstacle, which has hitherto resisted all mechanical pressure.

In that way, the perviousness of the air passages may be fully restored, and if all the exudation is expectorated, recovery is complete. But in many instances, the relief is only temporary. Either the process continues in forming fresh exudation; or the plug, or a portion of it, has only subsided from the centre to the periphery—generally towards the base, where it is comparatively innocuous. The obstruction, which it here produces, is, however, so slight that it is readily compensated. With the return of the plug to the centre, there is the return also of the asthmatic paroxysm, and this alternation of symptoms persists till the chest is entirely cleared.

TERMINOLOGY.

There is, apparently, some incongruity in applying the term "Bronchial Asthma" to the form of disease of which the nature has just been explained. It would, indeed, be desirable to abandon it altogether, and to substitute for it one, descriptive of the particular pathological process. But the question is, whether any such attempt will be successful. The term "Asthma" has, too long, been familiar to allow of any hope of its being displaced by another, however aptly it may be chosen. The one striking symptom—the paroxysmal dyspnœa—will continue to serve as the designation of the disease, especially as the absolute diagnosis depends, in some measure, upon microscopic examination. The introduction of a new name would only increase, instead of remedying, the confusion. There is, thus, merely a choice of evils. But though Asthma is misleading on account of the theories with which it is associated, it yet, as commonly understood, significantly expresses the clinical peculiarities of the affection.

In support of the continued application of the term, it may also be added, that the dyspnœal paroxysms, presenting the type above described, occur only in connection with fibrinous exudation, the tenacity of the sputa being essential for their production. In all other forms of bronchitis, the catarrhal and the purulent, the pathological products have so little cohesion, that they are readily expectorated, even when the energy of the muscles is considerably impaired. The larger tubes would be obstructed only in the case of an organic stenosis, in unconscious patients, and perhaps in adynamic fevers.

CHAPTER V.

ÆTIOLOGY OF BRONCHIAL ASTHMA.

A. Causes of the Disease: (*a*) Predisposing: Rickets and their Consequences; Disorders of Nutrition; Heredity; Age; Sex; Climatic Conditions; Antecedent Diseases; (*b*) Exciting: Micro-organism; Influence of Seasons.—B. Causes of the Paroxysm: Influence of Sleep; Forcible Respiratory Movements; Alleged Genius Loci; Alleged Alimentary Irritants.

A. CAUSES OF THE DISEASE.

(*a*) PREDISPOSING CAUSES.

A SPECIAL predisposition is at the root of Bronchial Asthma. Evidence of it lies in the fact, that the disease occurs only in a limited number of individuals, and in them, again, in peculiar but similar circumstances. Owing to the complexity of its nature, however, it is impossible to define, with any degree of precision, wherein it consists. Only an approximate insight into its working can be obtained by an analysis of the several factors which apparently contribute towards its origin.

Rickets and their Consequences.

The entire habit of asthmatics is, at once, indicative of certain constitutional defects. Rarely is a well-built and muscular individual found amongst them; and if there is one who, at first sight, may be taken to be perfectly healthy, a closer inspection reveals in him also characteristic abnormalities. The great majority of the patients have, indeed, been described as "miserable-looking wretches"[1]; and so far as their appearance is concerned the aptness of such a term, although by no means flattering, is nevertheless indisputable.

Amongst the one hundred and eighty cases, on the observation of which the following account of the ætiology is based, there were one hundred and forty-six—*i.e.*, 81 per cent.—in which the patients were either of stunted growth, or very tall and slim. All of them were in a state of marasmus, which was out of proportion to either the frequency, or the intensity of their paroxysms. In twenty-eight—*i.e.*, 15 per cent.—the average stature was nearly reached, or even exceeded. Here the predominant feature was obesity and great anæmia; while the tumid face and the thick layer of fat on thorax and abdomen strangely contrasted with the slender bones and the flaccid muscles of the extremities. In five only—*i.e.* 3 per cent. —the development was, apparently, normal; yet there was marked cyanosis, notwithstanding the absence of all appreciable diseases in lungs and heart.

In all asthmatics, without exception, the thorax is absolutely and relatively too small. It retains, in the adult, its infantile proportions, which are conspicuous enough in the emaciated, but are concealed in the obese. At the same time, there are a variety of deformities: the results on the one hand of kyphosis and lordosis, and on the other, of curvatures and infractions on the part of the ribs and the sternum. The barrel-shape, and pigeon breast are comparatively rare. Much more frequently, the chest is broad and flat, or represents the arc of a circle; or, by its angularities, resembles a square as nearly as possible.

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[1] Hyde Salter, *Op. cit.*, p. 401.

Though sufficiently striking, the so-called "asthmatic physique" has not received its due share of attention. Hyde Salter, indeed, noticed it, but mistook its bearing. He looked upon it as a complication, due to certain postures, and to the forcible contractions of the muscles during the dyspnœal attacks.[1] It is difficult, however, to conceive, how the mere mechanical agency, intermittent as it is and of short duration, could have any such effect. If the deformity appeared only in cases, in which the Asthma dated from earliest infancy, a supposed abnormal yielding of the bones would render that theory somewhat plausible. But, it is met with also in those, in which the paroxysmal dyspnœa first showed itself late in life, when ossification had long been complete; in which, moreover, there is abundant proof, that it had preceded the manifestation of the disease. Still less would the suggested cause explain the simultaneous presence of similar changes in other parts of the osseous framework —in the head as well as in the extremities. Nor would it account for the remarkable fact, that asthmatics, generally, belong to families, of which several members, though free from dyspnœa, are affected in a like manner.

No other conclusion, therefore, seems possible but that the deformities are the result of bygone rickets. From the histories of the cases nothing definite can be elicited in reference to any such infantile disorder. But that want of information on the subject is quite intelligible, considering the time and the circumstances at, and in which, the patients are generally seen. Besides, rhachitis, even in its severe form, presents, occasionally, such ambiguous symptoms, that its exact nature is not recognised. And it is still more likely to be overlooked, when it is limited to the thorax, its most favourite seat amongst all parts of the skeleton. But when, as in the present case, its stamp is so indelibly impressed upon the whole constitution, no special confirmation is needed.

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[1] *Op. cit.*, p. 154 *seq.*

Thus, rickets are, mainly, concerned in the disposition to Asthma. Although the pathological process is, ostensibly, localised in a number of bones; yet, occurring as it does, at a time of life, when development is most active, its consequences are, distinctly, perceptible throughout the entire organism. The enlargements of the spleen and of the liver testify to the profound disturbance which it produces. In many instances, recovery may, indeed, be, and possibly is, complete; but subsequent events abundantly show, that it was not so in the asthmatic.

The injuries which that disease inflicts are especially marked as regards the lungs. They are crippled, as it were, and are invariably below the average volume.[1] For, even if they followed, *pari passu*, the growth of the chest, hey cannot, in existing circumstances, attain their normal size. They must, inevitably, remain as small as is the cage, which contains them. But there are reasons for supposing, that also their formative energy may, occasionally, be impaired. So much, at all events, follows from some cases of Asthma, in which the existence of a typical emphysema can, in no way, be attributed to the operation of any of its commonly alleged causes. The emphysema is observed at the very first appearance of the dyspnœal paroxysms, and has not been preceded by cough or any other respiratory strain. Its origin is thus explicable only upon the assumption that, while on the recovery of the bones from their rickety changes, the expansion of the chest was progressing, the development of the lungs was arrested. Hence, their over-distension is the necessary consequence of a disproportion between the dimensions of the thoracic cavity and those of its contents.

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[1] Benecke, *Op. cit.*, p. 118.

Associated with the smallness of the lungs, there is, possibly, also a corresponding diminution in the calibres of the trachea and of the bronchi. Nothing is, as yet, definitely known on that point in reference to rickets. It cannot be doubtful, however, that the diameters of those tubes bear some relation to the volume of the organ, of which they form part. If so, the supply of air need not be materially lessened, thanks to the provisions for a suitable compensation. But the presence of pathological products in those narrow passages would, necessarily, create a considerable obstacle to the performance of the function, for which the means of adjustment are less readily available.

More important than their bulk is the state of their nutrition. It is hardly probable that, stunted as they are, they possess the average power of resistance to, and of repair after, injuries. In that case, the want of vitality will be the greatest in tissues, which depend for their nutriment, so to say, upon their neighbours. And indeed, the abundance, with which the epithelium was shown to exfoliate, is a proof of a but feeble cohesion with its matrix. With such peculiarities, there is necessarily an increased liability to all kinds of inflammatory changes.

Apparently, the heart escapes the injurious influence of rickets. Essentially a muscular organ, it is, on that account, in more favourable conditions with regard to growth, and less readily affected by the reduction of the capacity of the chest. In obedience to inherent and to external stimuli, it pursues its development at the expense of the more compressible lungs, appropriating, as it were, to itself all the needful space. In his anatomical investigations, Benecke[1] accordingly found it, as a rule, to be normal. Clinically, however, the question can rarely be decided; for the frequent complication with emphysema renders it impossible to determine the exact extent of the cardiac area.

In some cases, however, abnormalities may be suspected. Certainly, the size of the fist is no reliable standard. But there is this striking fact, that a number of asthmatics, perhaps the best-built amongst them, and even those accustomed to manual labour, have unusually small hands. At the same time, there is, generally, an amount of cyanosis, which is evidently not explained by the state of the lungs.

The frequency, with which enlargement of the thyroid is observed in Asthma, must be interpreted as an additional sign of a certain derangement, which takes place in the organism. Struma existed in fourteen out of the hundred and eighty cases—*i.e.*, in nearly 8 per cent. Five of them were those of males, and nine of females. The youngest patient was a boy, aged eleven. In three of them, again, there was slight exophthalmus; and in one, that of a female, aged twenty-one, it was reported that,

[1] *Op. cit.*, p. 252.

when thirteen years old, she had suffered a great deal from palpitation. In no case was there any sign of hypertrophy of the heart. None of the patients had ever been near a place, in which goitre is endemic. In two cases, mother and daughter were similarly affected. In all of them, its development was not attended by any special symptoms, nor was even its existence noticed till attention had been drawn to it. The gland caused no compression of the trachea, nor did its volume increase during the paroxysms. It had, certainly, no direct or indirect influence upon the production of the dyspnœa, as suggested by Schnitzler.[1]

Disorders of Nutrition.

An obstinate form of anæmia, which resists almost every remedial measure, is so characteristic of Asthma, that a large share in the disposition to the disease must be assigned to it. That it is not dependent on the frequency nor on the duration of the dyspnœa, is sufficiently obvious from its equal intensity in cases, in which the attacks are separated by long intervals of a complete freedom of respiration. It is, probably, only a part expression of rickets, and a clue to its origin and persistence may be found in the supposed intimate relation between the blood corpuscles and the bone-marrow. While it lasts, it certainly enhances the porosity of the vascular walls and favours the occurrence of fibrinous exudation.[2]

Heredity.

To avoid erroneous conclusions, which would necessarily arise from the elastic manner, in which the term Asthma is generally used by patients, only those cases, where the ascendants themselves were under observation, have been admitted into the following table :—

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[1] *Wiener Mediz. Presse*, 1877, *No.* 52.

[2] Heubner, *Die experimentelle Diphtherie*, 1883.

Disease.	Males.				Females.				Both Sexes.
	Gd. Prt.	Fath.	Moth.	Both Par.	Gd. P.	Fath.	Moth.	Both P.	
Asthma	4	5	6	1	2	6	4	2	30
Phthisis	—	1	6	2	—	2	3	—	14
Total	4	6	12	3	2	8	7	2	44

An hereditary element is, thus, strongly marked in thirty cases—*i.e.*, more than 16 per cent.—in which the parents themselves suffered from Asthma. Sixteen amongst them were those of males, and fourteen those of females. In the greater number of the males, the disease was transmitted on the mother's, and in that of the females, on the father's side. The rickety conformation, with all its consequences, was as distinct in the one as in the other generation.

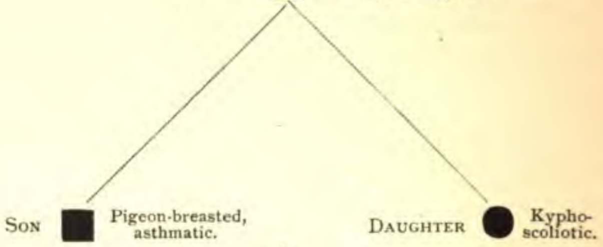
In fourteen cases—*i.e.*, nearly 8 per cent—in which nine were males and five females, there was phthisis in the ascendants. The greater influence of the mother was, here, also noticeable.

In many instances, the tendency to Asthma was attributed to carcinoma, and to general debility of the mother. But, as there was no opportunity of verifying these and similar statements, a detailed reference to them has been omitted. Certain it is, that the constitutional taint of asthmatics is frequently due to exhausting diseases of the parents.

In other cases, again, in which no reliable evidence of heredity could be obtained, several members of the same family suffered from Asthma.

In four cases, the disease was observed in one brother and one sister; in four cases, in two sisters; in two cases, in two brothers; in one case, in two brothers and a niece; in one case, in one brother, two sisters, and a nephew.

In still others, in which only one of a family was asthmatic, several of its members showed unmistakable signs of rickets, as in the following instance, which may be looked upon as somewhat typical.


FATHER
Short, stout, anæmic, square-shaped head, bow-legged.
SON
Pigeon-breasted, asthmatic.
DAUGHTER
Kypho-scoliotic.

Age.

The periods of life at which, in the cases observed, the disease has distinctly declared itself, were stated to be as follows:—

AGE.	MALES.		FEMALES.		BOTH SEXES.	
	No.	Per Cent.	No.	Per Cent.	No.	Per Cent.
Under 10	39	38·61	24	30·38	63	35·00
10—20	17	17·52	18	24·66	35	19·45
20—30	17	17·52	17	21·92	34	18·90
30—40	12	11·35	11	13·70	23	12·72
40—50	11	9·28	4	2·73	15	8·35
50—60	3	3·09	4	5·47	7	3·90
60—70	2	2·07	1	1·38	3	1·68
Total	101	100·00	79	100·00	180	100·00

With the greatest frequency Asthma develops itself in childhood. More than one-third of all the cases—sixty-three according to the above table—occurs in the first decade of life. The earliest ages, at which it was reported to have been recognised, were six months in three males; nine months in one female; eighteen months in one female and two males. Hyde Salter mentions one instance at fourteen days old, and another at twenty-eight.[1]

The infantile organism appears to be specially predisposed to fibrinous exudation. It is known, that children, above all the anæmic and the delicate, supply the greatest contingent to the epidemic diphtheria, as well as to the benignant forms of the disease. Even amidst the sputa, of what is supposed to be purely catarrhal bronchitis in them, pseudo-membranes are often found,[2] and their presence doubtless accounts for the intensity of the attendant dyspnœa. There is the record of a case, in which the air passages of a newly born infant were occluded by fibrinous coagula; so that the peculiar inflammatory process may, apparently, develop itself also during intra-uterine life.[3]

The tendency of children to that from of inflammation is readily accounted for by the greater softness of their tissues, especially that of the epithelium, and by the greater permeability of their vascular walls. In all probability, the leucocytes themselves are more prone to molecular changes, in virtue of which, they easily undergo coagulation necrosis.

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[1] *Op. cit.*, p. 112.

[2] Rilliet et Barthez, *Traité des Malad. des Enfants*, 1853, t. i., p. 410.

[3] Hayn, quoted by Lebert, *Klinik d. Brustkrankh.*, 1874, Bd. i., p. 112.

The physiological vulnerability, as it were, is necessarily intensified in Asthma, owing to the constitutional taint, which follows in the train of rickets.

The ages of between ten and twenty years supplied a contingent of thirty-five, and those of between twenty and thirty years, one of thirty-four cases. From their histories, so far as they could be ascertained, it seemed as if the dormant disposition had been roused by intercurrent diseases to which those ages are particularly liable; or as if the consequence of an arrest in the development of certain tissues or organs had become perceptible only when the growth was nearly complete.

Thus, in more than two-thirds of all the cases—in one hundred and thirty-two out of one hundred and eighty—Asthma arises before puberty. Thenceforward the tendency to it considerably abates; and as life advances its first manifestation becomes more and more rare. In only ten was it stated to have developed itself between the ages of fifty and seventy; and amongst them, were two females at 62 years, and one male at 65 years. It is evident, therefore, that the changes incidental to the normal involution of the body have little influence upon the disposition to the disease.

SEX.

The numerical preponderance is, decidedly, in favour of the male, of whom there were one hundred and one, to seventy-nine females. It is, however, too slight to allow any deduction as to the influence of sex. What has been said as to "men being more exposed than women to the inclemencies of the weather, to the wear-and-tear and hardships of life, and to the stress of violent and sustained respiratory efforts, as in heavy labour, and to intemperance —all these tending to the production of the disease"[1]— certainly does not hold in the present instance. The difference, as shown above, exists, in a marked degree only, in the first ten years of life, when the males are thirty-nine and the females twenty-four. Needless to say that, at that time, none of the alleged circumstances could, in any way, be operative. It merely coincides with the tendency of male children in general to all forms of disease. It disappears almost altogether from the tenth year onwards, when the sexes are nearly equally represented; the sole exception being between the ages of forty and fifty, when there were eleven men to four women—an exception, no doubt, purely accidental.

There is no evidence, that the performance of the sexual function in the female, both in normal and in morbid conditions, conduces to the origin of the so-called "uterine" Asthma. The tardy appearance of the catamenia, the menstrual molimina, the leucorrhœa, or similar affections, which are occasionally observed in those, who are subject also to that disease, are co-effects, not its causes. The disturbances on the part of the one and of the other organ primarily proceed from the arrest of development, which has above been insisted upon.

Pregnancy, on the contrary, seems to be in some way connected with it; but whether it is here, as in other forms of fibrinous bronchitis, accidental or causative, is difficult to decide. Amongst fifty females between the ages of twenty and fifty years, twenty-eight were married. Eleven of them were sterile, while five out of the remaining seventeen stated, that their Asthma had commenced during gestation. Towards term, they had been seized with pain in the limbs, shivering fits, severe coryza, cough, and then paroxysmal dyspnœa. In one case, the disease was supposed to have brought on premature labour, and in two cases the children were still-born. All the patients, however, had a marked constitutional and hereditary disposition, and were subject to severe sneezing fits also before pregnancy.

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[1] Hyde Salter, *Op. cit.*, p. 114.

Climatic Conditions.

Climatic conditions, in the general acceptation of the term, are not concerned in the disposition to Asthma. The stories, generally, current as to the injurious or favourable effect of the air of certain places have reference only to the attacks of the dyspnœa, not to the disease itself. On closer inquiry, however, those theories will be found to be devoid of all foundation. Many of the patients, that were observed, had lived and had equally suffered in almost all parts of the inhabited globe, and on land as well as on sea.

It is otherwise with climate, in its more restricted sense —the artificial atmosphere indoors—in which most individuals spend the greater part of their lives. Delicate from infancy, and generally the offspring of "delicate" parents, asthmatics guard themselves or are guarded with feverish care against the dreaded consequences of "catching colds"; and thus, in their anxiety to avoid a hypothetical danger, they expose themselves to a real one. The impurities of the air, which they are constantly breathing, impair the nutrition of the tissues and thereby increase the disposition to the disease.

Antecedent Diseases.

The first onset of Asthma has not yet been accurately observed. All patients were seen only, when they were confirmed sufferers from it. Therefore, the supposed influence of antecedent diseases upon it must for the present remain an open question. All that is known on the subject is derived merely from the reports of the patients or their friends.

Accordingly its origin has been attributed to the following:

To sneezing fits, colds, and hay fever	in 59	cases.
,, whooping cough and measles	,, 40	,,
,, diphtheria, croup, and tonsillitis	,, 14	,,
,, scarlatina	,, 4	,,
(In 44 of these cases eczema existed simultaneously.)		
,, bronchitis, congestion of lungs, and pneumonia	,, 38	,,
,, eczema, and other diseases of the skin	,, 7	,,
,, rheumatism	,, 2	,,
,, gout	,, 2	,,
,, syphilis	,, 2	,,
,, typhoid fever	,, 1	,,
,, general debility	,, 11	,,
Total	180	

Diseases of the Chest.—Admitting the accuracy of the diagnoses, it is certain only, that those affections preceded the appearance of the dyspnœal paroxysms. But there is no evidence, that they contributed towards their production. From the nature of the great majority of them—such as the various forms of coryza, of croupous exudation in the pharynx, and of the inflammatory changes of the air passages—it appears, with greatest probability, that they were already the first expression of the pathological process, of which the attacks of dyspnœa were merely a later manifestation.

Acute infectious diseases.—In forty out of one hundred and eighty cases, the dyspnœal paroxysms were said to have followed, either immediately or after a considerable interval of time, measles and whooping cough, both in their severe and their mild forms. In theory, it is quite admissible that these diseases may increase the disposition to Asthma; and the probability is even greater, if they are complicated by catarrhal pneumonia or by croupous exudations. Whether it is so in fact, there is as yet no means of saying.

As regards the frequency with which asthmatic paroxysms are preceded, as alleged, by whooping cough in children and in adults, it is but necessary to remember, that the form of cough during the prodromal stage of Asthma is, not infrequently, of a peculiar crowing kind,[1] differing from the typical pertussis only in not being contagious. It arises from the inflammation and from the œdema of the aryteno-epiglottic folds, which are sucked inwards on deep inspiration. It is said to be observable also in herpes of the pharynx.[2] It is, however, not a cause of Asthma, but merely an early symptom of the pathological process.

Diseases of the Kidneys.—The association of renal disease with Asthma is not very common. It was seen only in five out of one hundred and eighty cases. In two of them, it was more than probable, that the affection of the kidneys was the consequence of Asthma. In one, a case of granular atrophy, the first asthmatic paroxysm was reported to have occurred seven years before the fatal end. At the time, at which the patient was under observation, the return of the attack was in no way connected with uræmia. On the contrary, the quantity of urine and that of urea had increased rather than diminished immediately before and during the dyspnœa. There were signs, however, of indurative changes in the lungs, not only in that, but in every other case of chronic nephritis.

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[1] *Vide* p. 39.

[2] Trousseau, *Op. cit.*, t. i.; p. 332 *seq.*

In the case of a young lady, aged twenty-nine years, the Asthma even disappeared on the supervention of a renal complication. She had, since her eleventh year, suffered from a severe form of the disease every summer. Two years before her death, there was shrinking of the left side, with cavernous symptoms and displacement of the heart. Shortly afterwards, albumen was detected in the urine. In the last year of her life, when the kidney mischief had considerably advanced, she was quite free from dyspnœa.

Moreover, what is generally described as "uræmic" Asthma is not a definite form of disease, but one, that differs in its mode of origin and in its clinical peculiarities. That uræmic intoxication produces dyspnœa at all, has not yet been established. Still, acute and chronic Bright's disease are so frequently associated with fibrinous inflammation, that very probably they do give rise to the series of symptoms here under consideration.

Gout and Rheumatism.—The theory as to the origin of

Asthma from gout and rheumatism[1] is entirely based upon the occasional coincidence of those affections in the same individual. Nothing, however, warrants the assumption of G. de Mussy,[2] that in some gouty families, the diathesis may exclusively manifest itself by asthmatic paroxysms. Nor is there any proof that, because in two old women, the dyspnœal attacks were preceded by rheumatic pains in muscles or joints,[3] any relation existed between the several affections. So careful an observer as Dr. Garrod, and with his large experience of that class of diseases, does not support any such view.

Painful swellings of the joints were, however, observed in five out of one hundred and eighty cases. In one only were there tophaceous deposits in the ears; in the rest, the diagnosis of those symptoms remained doubtful. There was no family disposition, nor was there anything in the mode of life to point to gout. In all, there were not only pathological changes in the lungs, but the usual elements of the predisposition to Asthma.

Skin diseases.—Skin diseases are so frequently associated with Asthma, that probably some connection exists between them. The doctrines of humoral pathology assumed certain morbid principles in the blood, which affected the integuments and the lungs, and produced the variety of symptoms, according to their erratic localisations in the one organ or the other. That theory, to some extent, survives, and the alleged alternation between them is still maintained. The question, however, is as yet hardly ripe for decision. One difficulty in the way is, that skin diseases are much more prevalent than Asthma is, and that they, by preference, occur in the cachectic, such as asthmatics undoubtedly are. Another, equally great, is that their causes are still obscure. If bacteriological research, however, succeeds in confirming what at present must be looked upon as a mere matter of supposition, the cutaneous affections would, then, appear to be co-ordinate with Asthma.

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[1] Trousseau, *Loc. cit.*—Murchison, *Functional Derangement of the Liver.*—Garcia, *Asthme Goutteux; Thèse pour le Doctorat*, Paris, 1864.

[2] Lecorché, *Traité de la Goutte*, 1884, p. 310.

[3] Charcot, *Leçons Cliniques sur les Maladies des Vieillards*, 1874, p. 191.

In four cases, its origin was assigned to abscesses of the skin. The interest attached to them is, that the phlegmonous inflammation of the surface was apparently co-existent with an inflammatory affection of the pharynx.[1]

Mr. C., forty years old, with rickety-shaped head and flat thorax, was seized with "a severe cold"—*i.e.*, irritation of the nose and pharynx. These symptoms were soon joined by cough, which was particularly troublesome at night, and sounded like "whooping cough." A short time afterwards supervened a series of constitutional disturbances, which were supposed to be indicative of "typhoid fever." He was advised to keep his bed, and did so, till important business necessitated his immediate attention to it. To the consternation of his family and of his medical attendant, he got up. Throughout his illness there was a great deal of pain at the base of the chest. A circumscribed redness was then observed at either side of the junction of the seventh rib with the cartilage. The redness developed itself into an abscess, which burst and discharged a quantity of pus. Typical attacks of Asthma thereupon set in.

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[1] Conf. Gerhardt, *Berl. klin. Wochensch.*, 1887, No. 3.—A. Fraenkel, *Ueber septische Infection im Gefolge von Erkrankungen der Rachenorgane: Zeitsch. f. klin. Med.*, Bd. xiii., 1$^{\text{tes}}$H., 1887.

Mr. H., aged sixty-five years, of rhachitic conformation and in a state of great marasmus, had been subject to "colds" when a young man, and also to eczema of his hands. Eighteen years ago, his cough became very troublesome; he had at the same time, a sore throat and difficulty in swallowing. These symptoms subsided, but he continued to be feverish. A short time afterwards, a large abscess appeared on the right calf; then one, over the left olecranon; and a third, on the outer side of the right forearm. All of them had to be opened. On recovery from them, he became subject to Asthma.

Miss F., aged twenty-one years, with a history of phthisis on the father's side, of pronounced rickety constitution, very anæmic and emaciated, had from infancy, suffered from hay fever, and frequently from sore throats. She menstruated for the first time at nineteen. At first, the catamenia was attended with great pain, in consequence of which she underwent several operations on the womb. At eighteen years old, she was said to have had diphtheria, which was followed by an abscess on the dorsum of the right foot. Subsequently, she became asthmatic.

Mr. B., aged sixty-one, with a narrow rickety thorax, had, at thirty-one years old, a feverish illness, with sore throat and cough. An abscess then formed over the sacral region, and was opened by Sir Benjamin Brodie. The recovery from it took two years and nine months, and he then became asthmatic. Eighteen years ago Sir William Fergusson removed several polypi from his nose, but his Asthma was in no way benefited by the operation.

Syphilis.—In the two cases, a specific origin was alleged of the disease. Both patients, however, had hereditary dispositions, marked deformities of the chest, and histories of "hay fever" since infancy.

(*b*) EXCITING CAUSES.

The clinical peculiarities of Bronchial Asthma plainly indicate the nature of their exciting cause. As has above been shown, the pathological process involved is a progressive form of inflammation. Starting, as it invariably does, from the pharynx, it rapidly spreads upwards to the nose and to the eyes; whereas its downward extension is, for a time at least, arrested by the larynx, just as erysipelas is arrested by a projecting fold of skin. That resistance, however, is subsequently overcome, and the air passages are then invaded. Gradually also the tissues are structurally altered, and their reaction differs. The sero-fibrinous exudation becomes more and more fibrinous, where such is wont to occur; and the consequent mechanical interference with the respiratory function now constitutes the most striking of all its symptoms.

No mechanical nor chemical irritant—no foreign body, no abnormally high nor abnormally low temperature, no vaso-motor neurosis, nor anything else, that has hitherto been alleged as a provocative of the disease—can possibly give rise to such a series of phenomena. None of them can exert its injurious influence beyond its immediate point of impact. But in Asthma, the one characteristic feature of the morbid changes is their steady creeping along the surface, and their tendency to the formation of a specific product.

The agent, endowed with such properties, must necessarily be one, that is capable of reproducing itself. Suspicion, therefore, attaches itself to the various micro-organisms, which have previously been described.[1] The most constant amongst them was, as will be remembered, a streptococcus (Plate VI., Fig. 27). It was invariably found amidst the large and the small cords, and more abundantly so, in the recent than in the old. It was seen on the surface of the smallest bronchi (Plate VII., Figs. 33 and 34), and between the fibrinous exudation into the infundibula (Plate VI., Fig. 32). The bacillus (Plate V., Fig. 26) was only occasionally met with, and the capsule-coccus (Plate III., Fig. 12) exclusively occurred in the pseudo-membranes.

It shall, at once, be conceded, that the mere presence of a micro-organism, however constant it may be, proves absolutely nothing as regards its pathogenic nature. Nor would it serve any useful purpose to refer here to the failures, which attended my numerous attempts to satisfy, as far as possible, the postulates of bacteriological science, in order to arrive at some decision on that point. What follows is admittedly only hypothesis; but hypothesis which, it appears, is not altogether unfounded.

A variety of circumstances render it highly probable, that a streptococcus is the cause of the progressive form of inflammation, peculiar to Bronchial Asthma.

In the first place, it has been found also in the croupous exudations, complicating measles and whooping cough,[2] and in a case of idiopathic fibrinous bronchitis.[3]

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[1] *Vide* p. 59.

[2] Thaon, *Revue de Méd.*, 1885, p. 1017 *seq.*

[3] A. Fraenkel, *Charité Annal.* xi. *Jahrg.*, pp. 196—204.

Secondly. The most distinctive characteristic of its action is the tendency to spread along the surface, as it is observable in erysipelas of the skin.

Thirdly. Consequences, closely similar to those seen in Asthma, have been assigned to its influence by the most competent observers. Weichselbaum[1] maintains, that it produces a form of pneumonia, and a sero-fibrinous exudation into the connective tissue from the nasal cavities down to the hilus of the lungs.

Löffler also considered it to be the cause of the disease in the following case:—

"A child at the breast, eight months old, was seized with running from the nose, and on examination there was exudation in the pharynx. Seven days later, dyspnœa set in. There were slight dulness and rhonchi on the right side behind. Tracheotomy was performed, and on the discharge of a few membranes, there was a temporary improvement. Death, however, ensued three days after the operation."

"*Post mortem.* No exudation upon the tonsils, and but scanty fibrinous masses in trachea and bronchi. Broncho-pneumonia of lower portions of the lungs.

"The epithelium of the epiglottis was intact. The lymphatic vessels contained dense masses of streptococcus.

"The epithelium of the trachea was destroyed. On the surface was a thin layer of cellular exudation, with abundance of streptococcus, which penetrated into the mucous membrane itself. No other form of bacteria was present.

"The alveoli were, here and there, filled with cells and fibrine. The bloodvessels were greatly dilated and full; and there were hæmorrhage and exudation. Some of the perivascular lymph-spaces contained swarms of streptococcus, which ran through the contents of the alveoli.

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[1] "Ueber Aetiolog. u. Pathol. Anatom. der Acut. Lungenentzund." *Wien. Med. Woch.*, 1886, No. 39 *seq.*

"Heart, liver, spleen and kidneys were completely free from bacteria."[1]

All that has just been mentioned, applies, however, mainly to the streptococcus erysipelatos. Against it is the fact, that a similar form of bacterium is said to be a harmless inhabitant of the mouth in health. Still, the rigid barrier between the one and the other does apparently not exist. There is the possibility, that the purely saprophytic organism may acquire pathogenic properties, either by a rapid numerical increase, or by a diminution in the resistance of the part of the host, or by a combination of both these circumstances. Baumgarten[2] contends that they agree not only in forms, but in their habits of life as ascertained by cultivation; and further, that the presence of decomposing organic matter—as for instance in erysipelas—is most favourable for their development. Hence he concludes, that the streptococcus, which is found in the normal secretion of the vagina, may become virulent, and produce diphtheritic endometritis during the puerperal state, if its access to the injured lining of the uterus is not impeded; and that the otherwise innocuous streptococcus of the mouth may give rise in scarlet fever and measles to the frequent complications with diphtheria.[3]

Nowhere are conditions so favourable for the acquisition, as well as for the exertion, of such aggressive influence, as in the disposition, that tends to Asthma. The emigration of leucocytes from between the adenoid tissue of the pharynx[1] is here much more abundant than normally. In the organic matter, thus supplied, the streptococcus, favoured by the higher temperature of the mouth, finds a ready nidus for its proliferation; and its subsequent invasion meets with but little resistance on the part of the yielding epithelium.

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[1] Löffler, "Mittheilungen aus dem Kaiserl. Gesundheits-Amte," ii. Bd., 1884, p. 449.

[2] *Lehrb. d. Patholog. Mykologie*, 1887, p. 235.

[3] *Op. cit.*, p. 347 *seq.*

Influence of Seasons.

Asthma is, undoubtedly, more frequent in the summer than at any other time of the year. If the streptococcus may be assumed to be its exciting cause, its prevalence during the hot weather admits of a satisfactory explanation. For, on the one hand, there is, owing to the dilatation of the vessels from the heat, a greater liability of the respiratory surface to inflammatory changes. On the other, the higher temperature is the optimum for the growth of the microbe, which obtains its greater virulence with its state of maturity. Besides, in the hot and dry weather the inspiratory current is full of particles which, by the force of the wind, are lifted into the atmosphere from the superficial layers of the soil; and if it happens to contain, as it frequently does, the particular fungus, an infection is at once produced in a predisposed individual.

There is no foundation for the current theory, that the abortive form of Asthma, commonly known as "hay-fever" or "hay-asthma," is due to the inhalation of the pollen of certain grasses. It is based entirely on misconceptions and erroneous inferences. Bostock, as is well known, was the first to draw attention, in a report of his own case, to a severe form of coryza which occurred during the heat of summer;—at a time of the year, when it could not be traced, by any stretch of imagination, to its only recognised cause—cold. The most probable ætiological factor, that suggested itself to him, was, therefore, the heat.

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[1] Ph. Stöhr, "Zur Physiol. d. Tonsillen," *Biolog. Centralbl.*, Bd. ii., No. 12, 1882.

The facts, as stated by him, were soon confirmed; but their alleged cause was contested. At first, the symptoms were attributed to the odour, and now, they are almost unanimously admitted to arise from the inhalation of the pollen, of certain grasses.

Yet in the series of symptoms, supposed to be peculiar to hay fever, there is absolutely nothing, which distinguishes it from any other form of coryza, such as occurs at all times of the year, in summer as well as in winter; either as an idiopathic affection, or as a part expression of certain acute infectious diseases.

Its sole claim to individuality rests on its assumed mode of origin, and, it must be added, on the accretion of a number of stories around it. The one is, that the educated, or rather the well-to-do, are chiefly subject to the disease. But, then, it must not be forgotten, that these are conversant, if not with a special kind of literature on the subject, at all events with some of the current theories of medicine; whereas the poor have no difficulty in persuading themselves that, what they are suffering from, is merely a "cold," which is patiently borne as the inevitable lot of humanity.

Nor is it quite correct, that those, subject to hay fever, are altogether exempt from "colds" in the winter. Their statements on that point are not reliable. One lady positively declared, that such was the case with her; but, in the course of conversation, she related how, curiously enough, she had been seized one night in November, whilst lying in bed, with one of her typical attacks.

Given opportunities of closely observing them, it will be found, that they suffer from coryza also in the midst of winter, even when confined to their rooms for weeks. Only in such cases the diagnosis is somewhat arbitrary. Thus, one lady had all those characteristic symptoms early in the month of April. Her medical attendant, at first, suspected that she had somewhere caught the measles; but she herself recognised in them her habitual "hay fever."

As a matter of fact, however, all forms of coryza and of angina are, upon the whole, more prevalent in the summer, than in the winter. The general opinion to the contrary is merely the consequence of rashly identifying the sneezing fits, which arise from the passage of cold air over an over-heated nasal mucous membrane, with those, that are a symptom of a peculiar inflammatory process.

Doubtless pollen, like any other foreign body, produces sneezing if inhaled through the nose. But it is not, and cannot be, the cause of hay fever. If it were so, it would be quite inconceivable why, as generally admitted, the affection should invariably start from the back of the palate, should then spread upwards to the nose, and also to the eyes[1]; considering that these parts are, first of all, exposed to its alleged pathogenic properties. Nor would it be intelligible, why individuals should suffer, as they certainly do, for weeks after they have been subtracted from its supposed influence, as the pollen would be readily washed off the surface by the copious stream of exudation, which its presence provokes.

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[1] Blackley, *Experimental Researches on the Causes and Nature of Catarrhus Æstivus*, 1873, p. 171.

What is called hay fever is, in reality, the abortive form of Asthma, and due to the same predisposing and to the same exciting causes.

In twelve typical cases of the disease, the discharge from the nose, and small portions of epithelium, scraped off the pharynx, the root of tongue and the nasal mucous membrane by means of a sterilised platinum wire, were submitted to examination in the usual way. Abundant masses of the streptococcus were found lying between the epithelium, except in that, coming from the nose. The forms of bacteria, which are generally found in the mouth, were absent.

"Cold" is frequently accused as the cause of Asthma, especially by patients who least expose themselves to its action. There are some who, on the contrary, emphatically declare that they are always better in the winter, and who even hail the dreaded east wind and the fog as sources of relief.

Cold has no necessary connection with the occurrence of the disease in the winter, nor with the production of the bronchitis, which is supposed to be the intermediate link. The wide-spread notion of its injuriousness has probably originated in the observation, that almost all acute affections are ushered in by chills or rigors, which often for days precede their local manifestations. Thus, what is actually their first expression is, erroneously, considered to be their cause. A great deal of ingenuity has been expended upon experiments, to establish that theory upon a scientific basis. Hitherto they have not led to the desired results. Rossbach,[1] however, claims to have solved the question. The application of an ice-bag to the abdomen of a small animal was seen to somewhat increase the secretion of the glands in the tracheal mucous membrane. However this may be, the conditions in which the observations were made, are widely different from what is generally understood by catching cold. Daily experience proves, that even an ice-bag on the abdomen produces no catarrh in man.

ÆTIOLOGY OF THE PAROXYSMS.

Thrombosis.

The inflammatory process is occasionally so intense in the large bronchi, that a considerable number of them is obstructed by the exudation. The alternate formation and removal of the fibrinous casts, sufficiently, account for the varying intensity of the dyspnœa. Here, therefore, the causes of the disease are, at the same time, the causes of the paroxysms.

Embolism.

Oftener the dyspnœal paroxysm is of embolic origin. The exudation formed in the smaller tubes is, after its detachment from its matrix, rolled up; and in its onward course it is, owing to its great tenacity, its shape and size, arrested in a main bronchus.

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[1] "Ueb. d. Schleimbildung u. die Behandlung d. Schleimhaut-Erkrankungen in d. Luftwegen," *Festschrift der Alma Julia Maxiliana*, Leipzig, 1882, Bd. i., p. 85 seq.

Influence of Sleep.

A variety of circumstances contribute towards the frequent occurrence of the attacks during sleep. Neither the night, as such, nor the darkness,[1] as alleged, has anything to do with it. All depends upon the state of the patient, whilst he is awake. If his smaller tubes, then, happen to contain sufficiently movable plugs, the main passages are likely to be obstructed. There is, probably, a greater disposition to the dyspnœa, owing to the physiological reduction in the interchange of gases during sleep, and to the consequently greater irritability of the respiratory centre. But the most important factor is, undoubtedly, the recumbent posture. Along the inclined plane, into which the surface of the bronchi is thus converted, the progress of the exudation from the periphery toward the centre is greatly facilitated; and even the shallow movements of asthmatics, then, succeed in effecting what they fail to do in the erect position, when the direction is nearly perpendicular. Possibly, also, the restless tossing in bed under the influence of disquieting dreams[2] may break up a thrombus and cause impaction of its fragments.

Forcible Respiratory Movements.

The seemingly fitful appearance and cessation of the dyspnœa admit of a rational interpretation, if it be but remembered, that asthmatics enjoy their comparative freedom of breathing, thanks to certain compensatory arrangements. The persistent reduction of their respiratory surface, in consequence of the obstruction of the bronchi, has led to such a decrease of their respiratory wants, as they are able to supply without conscious effort by means of shallow respiratory movements. Hence, so long as the demands remain within certain limits, the plugs are not disturbed; for the inspiratory current is too feeble to displace them, and they are subject only to the inevitable process of decay.

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[1] Lebert, *Loc. cit.*, p. 445.

[2] Hyde Salter, *Op. cit.*, p. 70.

Once, by some means, detached from its matrix, the exudation gradually moves upward, and the ventilation of the lungs becomes still more imperfect. The result is, in many instances, especially in debilitated individuals, an irresistible state of drowsiness and languor; in others, again, a profuse diuresis;—all of which are recognised as the signs of an approaching attack.

At times, however, the exudation, although still firmly impacted, may nevertheless be, suddenly, detached by forcible respiratory movements: such as laughing, coughing, and sneezing. In the midst, then, of what appears to be a state of perfect health, an attack of Asthma takes place. It is not generally possible to trace the sequence of events, because the patients are rarely under constant observation. But where the opportunity is given, it will be found that those expiratory acts are followed by stenotic rhonchi all over the chest, where previously the sole abnormality, that could be detected, was either complete silence or only a peculiar soft breathing over some part of the lungs. The effect of cough was strikingly shown in the following case:—

Mr. X., æt. 62, subject to Asthma fifteen years since, was seen one morning in August, when he declared himself to be PERFECTLY WELL. Indeed, except complete silence from the right nipple downwards, the physical signs were negative. He was directed to cough repeatedly, and immediately afterwards, respiration disappeared from under the right clavicle, where, but a few minutes before, it was most distinctly heard. At the same time, distant rhonchus was faintly discernible at the right base in front. He attended to his business during the day—not, however, without some discomfort. But he was obliged to return home early in the evening, on account of a great drowsiness. He actually fell asleep over his dinner, and slept on in his chair till about ten o'clock p.m., when he awoke with a sharp attack of Asthma.

He was seen one hour later, when the following was noticed. Respiration 18. Prolonged expiration; inverse action of diaphragm. Distension of the chest. Absence of vocal fremitus on both sides. In front, tympanitic, and on both sides behind, slightly impaired resonance. Tracheal wheezing. Complete silence on both sides in front, and on the right side behind. Feeble respiration and cooing left side behind. He expectorated a quantity of casts, whereupon the dyspnœa subsided and the chest cleared up.

Thus, the dyspnœa, which is said to arise[1] from the inhalation of a variety of foreign bodies suspended in the air, is due to the sneezing produced by them. The best known in that respect is powdered ipecacuanha. It is evident that the result can, to some extent, be obviated by plugging the nostrils,[2] or by painting the nasal mucous membrane with cocaine.[3] In the one case, the irritation is not caused by breathing through the mouth; in the other, its manifestation is prevented.

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[1] Blackley, *Op. cit.*, p. 58.—Cook and Berkeley, *Fungi*, 1875, p. 216.—Reyher, "Ueber gewisse Schaedlichkeiten in der Luft v. Wohnraumen," *Petersb. Med. Woch.*, Nos. 20 and 21, 1878.—E. Lewy, *Seltene Formen. v. Bleivergift. Oest. Zeitsch f. prakt. Heilk.*, 1870.

[2] Gottstein, *Breslauer aerztl. Zeitsch.*, 1881, No. 15.

[3] Hofmann, *Bericht d. laryngol.*, Section d. 57ten *Naturforscherversammlung*, Magdeburg, 1884.

By its influence on the respiratory movements, emotion also may provoke an attack, and there are reports of numerous instances, in which it is said to have done so.[1] That it occasionally cures[2] Asthma—so far, at all events, as the relief of the dyspnœa goes—is by no means surprising, for the force which is capable of impacting a plug may succeed in entirely removing it.

Alleged Genius Loci.

Every asthmatic asserts, that there are some places, where he is especially prone to suffer; and that there are others, where he is practically cured from his dyspnœa. Their opinion on the influence of locality, and the far-reaching inferences therefrom, appear to be nothing but hasty generalisations from isolated facts, which are, by no means, always reliable. Thus, one lady lamented, that her attack was due to the clay soil of the house, at which she visited at the time. Clay soil, she maintained, invariably, produced Asthma in her, whereas gravel always relieved it. Actually the house was on gravel. She was persuaded, however, to move to one, which did stand on clay; there she made a good recovery, and was for some time free from dyspnœa. A clergyman had a paroxysm in an hotel in Paris, and forthwith he attributed it to his bedroom facing south. He, too, was convinced, that a southern aspect regularly provoked his Asthma. But when the landlord was summoned to make the necessary arrangements for removal into another room, he had no difficulty in pointing out, that the impeached one was really to the north.

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[1] Hyde Salter, *Op. cit.*, p. 125 seq.

[2] *Ibid.*, p. 210.

Indeed, there is little foundation for all the current stories. It is obviously inadmissible to arraign the air of certain places, when there are numerous other circumstances, that are more likely concerned in the production of the dyspnœa. This much, however, is undeniable: that asthmatics frequently suffer shortly after their arrival at the seaside, or in the country. Such occurrences, however, do not justify the conclusion that "the worse the air for general health, the better, as a rule, for Asthma," and that "the worst parts of cities are the best, and conversely."[1] The origin of the dyspnœa, in those circumstances, is due to the vivifying influence of the bracing air, which increases the depth of breathing, and thereby tends to displace and impact plugs, which were not displaced by the shallow respiration in the vitiated atmosphere. The ensuing attack is painful; but it is, notwithstanding, a salutary effort.

Alleged Alimentary Irritants.

Equally erroneous is the theory as to certain articles of diet being "highly asthmatic."[2] Not the quality of the food, but its quantity, is apt to produce dyspnœa. The respiratory wants are by 20 per cent. greater shortly after, than before meals; and the forcible movements in order to supply the increased demand may cause either a bronchial obstruction, or, as it more frequently happens, merely asthmatic wheezing.

The flatulence, which almost always exists at the end of an attack, is the consequence, not its cause. Air is sucked into the alimentary canal, when, as is generally the case at the time, the patient breathes through the mouth. Intense tympanitis, however, especially when the diaphragm is more yielding than the abdominal muscles are, is apt to induce an intense form of dyspnœa.

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[1] Hyde Salter, *Op. cit.*, p. 300.

[2] *Ibid.*, p. 262 seq.

CHAPTER VI.

COMPLICATIONS AND SEQUELÆ OF BRONCHIAL ASTHMA.

Productive and Atrophic Changes of the Nasal Mucous Membrane: Hyperplastic Catarrh and Polypi; Atrophic Catarrh; Epistaxis; Olfactory Hyperæsthesia—Diseases of the Buccal and Pharyngeal Cavities: Croupous Exudation on the Tongue; Productive and Atrophic Changes in the Pharynx, Uvula, and Tonsils—Chronic Œdema of the Larynx—Diseases of the Lungs: Bronchiectasis and Emphysema; Cirrhosis; Broncho-pneumonia; Croupous Pneumonia; Rapid Phthisis—Diseases of the Heart: Hypertrophy and Dilatation of the Right Ventricle; Valvular Lesions; Functional Derangement—Disturbances of Digestive Organs — Diseases of the Kidneys — Gout and Rheumatism — Diseases of the Skin—Nervous Disturbances —General Nutrition—Coincidence with Acute Diseases.

ANATOMICAL lesions and functional derangements of various kinds are met with in almost every case of Asthma. Some of them are, manifestly, the inevitable consequences of the disease; others are nothing but purely accidental complications; and still others are said to, and probably do, stand in a generic relation to it. But this question is not yet ripe for decision; for, though some of those affections precede the appearance of the paroxysmal dyspnœa, there is no proof that they have caused it. At all events, there is, till now, no clue as to the connecting link between them.

PRODUCTIVE AND ATROPHIC CHANGES OF THE NASAL MUCOUS MEMBRANE.

Hyperplastic Catarrh and Polypi.

In more than two-thirds of all the cases, the repeated return of the erysipelatous form of coryza leads, in the course of time, to a hyperplasia of the nasal mucous membrane. The process is, as a rule, diffuse, and then chiefly manifests itself in the vascular tissues of the lower turbinated bones. Occasionally, however—in eleven out of one hundred and eighty cases—it is more circumscribed and also more active, resulting in the formation of what are commonly called polypi.

Those changes are attended by considerable inconveniences. In the first place, they necessarily narrow, or, according to their extent, entirely occlude the nasal cavities. Much, in that respect, depends, however, upon the temporary state of the fulness of the vessels: but as they are, here, more yielding than normally, they are therefore more liable to congestion from all kinds of vaso-motor disturbances, for which opportunities are hardly ever wanting. Thus, the obstruction is generally complete and permanent on both sides. At times it affects only one of them, and then often rapidly passes to the other, greatly to the surprise of the patients.

The consequences are, that respiration must be carried on through the mouth. The supply of air to the lungs is therefore reduced; for a great part of it follows the direction of the channel and gets into the alimentary tube. So also the inspiratory current is neither warmed nor filtered, although the loss of these functions of the nose is, by no means, so injurious as is commonly believed.

Of much more serious import is the retention of the abnormal secretion behind the obstruction. Masses of decomposing mucus accumulate in such cases within the retro-nasal space, and are often seen to project beyond the velum palati. In the recumbent posture, especially during sleep, portions of them fall down, as it were, upon the larynx, and their sonorous vibrations then produce the wheezing which is so alarming to the friends of the patient, but is hardly perceived by himself. Frequently they are sucked into the air passages, and unless they are speedily expectorated, they are apt to set up inflammation.

Again, the hyperplastic catarrh is the cause of the severe fits of sneezing—more than ten or twenty times in rapid succession—from which asthmatics generally suffer in the morning. Owing to the altered state of the vascular walls, the transudation into the surrounding tissues is considerably increased, especially during sleep, and the infiltrated fluid which the veins and lymphatics are unable to remove, excites, on waking, those explosive expiratory acts.

Atrophic Catarrh.

In other cases, the exudation into the nasal mucous membrane rapidly undergoes regressive changes, and the glandular and other tissues become involved in the process. The result is a more or less complete atrophy. The nasal passages are then wide and pervious. Their lining has lost its anatomical peculiarities, and resembles a serous, rather than a mucous surface. Sneezing fits rarely occur in these circumstances; more frequently, even in the acute exacerbations of the disease, there is a copious watery discharge which trickles down from the nose in large drops.

In seven of these cases, there was considerable ozæna, which, with two exceptions, readily yielded to simple treatment. In some of them, it appeared as if the increase of the ozæna was soon followed by a return of an acute attack of Asthma.

Epistaxis.

Epistaxis is, upon the whole, rare, and generally slight. It takes place both during, as well as in the intervals between, the attacks. In one case, in which it was very abundant, it was connected with granular atrophy of the kidneys, but it never occurred during the dyspnœal paroxysms.

Olfactory Hyperæsthesia.

A certain repugnance to the scent of flowers was alleged to exist in one case. The patient, a lady, declared that their presence affected her injuriously. That it did so, however, was never observed. She, who at one time, would loudly call for the instant removal of those objects, as being oppressive to her breathing, would at another, just when she was suffering from Asthma, have her forehead bathed with eau de Cologne. Or, in the intervals between her attacks, she would freely use toilet vinegar and highly scented hair washes, etc., without perceiving a return or an aggravation of her symptoms. Hyde Salter[1] relates a number of curious cases, in which there seemed to be some hyperæsthesia to the emanations from various animals. What is said of "rose colds," of the sight of a well painted hayfield,[1] or of even an artificial rose, producing Asthma, admits, perhaps, of a different interpretation from what is generally put upon it.

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[1] *Op. cit.*, p. 140 *seq.*

DISEASES OF THE BUCCAL AND PHARYNGEAL CAVITIES.

Croupous Exudation on the Tongue.

In the acute attacks of the disease, a certain amount of exudation invariably takes place on the tongue, especially near its root; but it is seldom of such an extent as to attract particular attention. In five cases, however,—three of them those of females and two of males—it was very abundant, forming a continuous coating of a milk-white colour, and several lines thick. It covered the surface of the organ from the tip to the root, and was visible also on the corresponding part of the pharynx.

On microscopic examination, it was found to consist of epithelium, which had undergone Wagnerian degeneration; of masses of micro-organisms; and of a network of long hyaline fibres, between which cellular elements were embedded, but it contained no thrush fungus.

Beneath it, the mucous membrane of the tongue looked glossy, and the fur always reappeared a few hours after removal. Apparently, it extended also down the œsophagus, for the patients complained of a feeling of obstruction, as if something occluded the passage. There was some dysphagia, which generally occurred between meals, when the organ had rested for a time, so that solids could only be swallowed with the help of water.

The exudation, as a rule, subsided on the cessation of the acute symptoms. But in the following case, it appeared to be persistent, and obstinately resisted every treatment.

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[1] Phœbus, *Op. cit.*, p. 30, § 11.

Mrs. X., forty-two years old, with a history of phthisis on the mother's side, had been delicate from infancy, and round-shouldered as long as she could remember. As a child, she had suffered a great deal from sore throats; and at the age of seventeen, she had had a severe attack of quinsy, on account of which, she had been leeched and blistered. At the time, her sputa had been streaked with blood. Menstruation had commenced at the age of fifteen, and then was regular, rather copious, but painless. She married at nineteen; four months afterwards she had a severe attack of bronchitis, which brought on a miscarriage. She was supposed to have recovered from the bronchitis; but, henceforth, she became easily out of breath, so that she deliberately avoided every exertion, and consequently began to get stout. With her last and sixth confinement, about ten years ago, she is said to have had pyæmia and abscess of the liver, which had burst through the lungs. Since then, she has been subject to typical attacks of Asthma.

At first, her attacks came on once a month; then, once every five or six weeks, or even every three months. But for the last two years, she has hardly ever been free from great oppression at the chest, and has been obliged to smoke a certain kind of cigarettes several times a day. Her dyspnœal paroxysms are, as a rule, preceded by a feeling of itching, extending from throat to ears, so that occasionally she is quite deaf. There is also pain between the shoulder-blades, more especially towards the right side. She has very little cough, and the sputa are of a greyish-white colour, and very tenacious. She has particularly noticed, that when sitting still, she had a sensation as if her gullet were obstructed, so that she felt inclined to try and remove the obstacle by eating a biscuit; but she always found that she was unable to swallow without drinking some water.

Stat. Pres.—Middle stature. Rickety shape of head. Great pallor, and slight cyanosis. Considerable obesity. Res. 24, noisy. Pulse 78, regular, very small and soft. Thorax barrel-shaped, marked kyphosis from the fourth cervical vertebra downwards. Hyper-resonance in front and behind on both sides. Respiration feeble; dry rhonchi all over, especially both bases behind. Cardiac impulse not seen nor felt. Cardiac area cannot be made out. Action of the heart regular, and sounds clear and feeble. Teeth healthy, but molars wanting. Tongue large, flabby, covered with a thick, snowy-white fur, several lines in thickness, in appearance like coagulated milk. Patient states, that if she scraped her tongue, the fur would return within a few hours. Chronic pharyngitis. Appetite good; bowels regular, but some flatulence. Liver not enlarged. She passes little water, her ankles occasionally swell, but there is no albumen in her urine.

She was seen again a few days later. She complained of general malaise, and had had a slight rigor. The temperature was 101·5° F., and she looked paler even than usual. The pharynx was dark red and glossy; she had no pain in the throat, and no appetite. Her bowels had freely acted. She had had several restless nights, but no severe dyspnœa.

In the following night, an attack of Asthma came on. When seen early in the morning, her face was flushed and bathed with perspiration, and she was leaning with her elbows on her knees. Respiration 18, with prolonged expiration. Pulse 120; very small, soft, and paradoxical. Her respiration was peculiarly noisy. Standing by her bedside, it seemed to me, as if a child were crying in an adjoining room. On listening, however, to the larynx and to the upper cervical vertebræ, the same kind of noise was heard very loud, and was conducted also all over the chest. The thick and white fur previously mentioned coated the tongue, on the depression of which, it was seen to be continued to the opposite surface of the pharynx, the distance between these parts having apparently been reduced by the compensatory lordosis of the cervical spine. At the pharynx was a semicircular diaphragm with fimbriated edges, stretched over the larynx, and its sonorous vibrations produced the peculiar sounds. The microscopic examination of the membrane showed its composition to be as above described.

On the removal of the diaphragm, the noise ceased. Rhonchi were then heard all over the chest. The sputa were about two tablespoonfuls, pearly white, tenacious, and of the usual character.

Productive and Atrophic Changes in the Pharynx, Uvula, and Tonsils.

The tissues of the pharyngeal cavity present the same changes as those, that take place in the mucous membrane of the nose. Atrophy, however, is in them decidedly preponderating, and it is especially remarkable for its occurrence even in very young asthmatics. It is a sign of their premature senescence, and connected with their general marasmus. In many cases, the sensibility of the pharynx is diminished. The application of instruments or of a penholder, for instance, produces no contraction of the subjacent muscles.

In others, again, there is a very distressing feeling of nausea and faintness, which is probably caused by the tenacious exudation, firmly adhering to the mucous membrane.

CHRONIC ŒDEMA OF THE LARYNX.

In consequence of the wasting of its tissues, the larynx is frequently the seat of chronic œdema. Parts of its tumid mucous membrane may occasionally be sucked into the glottis, especially on the deep inspiration during cough,[1] which thus obtains a close resemblance to pertussis. The glottis may in that way be entirely closed, as was seen in the following case:—

Mrs. H., aged forty-five, and subject for more than two years to typical attacks of Bronchial Asthma, complained that she had had several fits, and had even fallen down in the street. Nothing was known of their nature, nor could anything be conjectured as to their origin.

One day, however, she was seized, in the course of conversation, with cough, which in her case was frequently accompanied by long-drawn stenotic inspirations—distinct whoops. Suddenly, respiration entirely stopped; she became unconscious, and fell forward. The face and neck were swollen, and of a deep coppery colour, as if she had been strangulated. The jaw dropped, and saliva oozed from her mouth. There was no pulse at the wrist. The attack could not have lasted more than one minute. On recovery, she moaned and seemed dazed, asking, in a frightened manner, what had happened. Recollecting, as it were, the peculiar sensation in the larynx, she put her hand to it, to ascertain whether the obstruction had been removed. She complained of pains across the temples and in the cardiac region; but they soon passed off, and in less than ten minutes, everything returned to its former state.

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[1] Middeldorf, *Galvano-Kaustik.*, 1824, p. 208, quoted by Eppinger, *Patholog. Anatomie d. Larynx u. d. Trachea.* Berlin, 1880, pp. 27 and 66.

DISEASES OF THE LUNGS.

Bronchiectasis and Emphysema.

The obstruction of a certain number of air tubes, and the consequent collapse of the corresponding portions of the alveoli, are necessarily attended by a complementary distension of those parts which still remain pervious. Such forms of bronchial and alveolar dilatations often arise acutely, and may entirely subside, on the cessation of their cause.

It is doubtful, however, whether, in the protracted forms of Asthma, the recovery is ever complete. If the exudation is indefinitely retained within the bronchi, the emphysema and the bronchiectasis must also become permanent, and anatomical changes inevitably follow. Satisfactory evidence of their presence is, however, rarely obtainable by means of physical signs. The more or less extensive atelectasis deprives the emphysema of its characteristic signs—over-distension and hyper-resonance; while, even in the most favourable circumstances, bronchiectasis can seldom be recognised by means of auscultation and percussion. But, there are the ætiological factor, and more especially, the great quantity of sputa, which render the diagnosis highly probable.

Cirrhosis.

In five cases,—four females and one male,—there was considerable induration with shrinking of one lung. The disease was observed only after its complete development, and nothing could be ascertained of its origin and progress. In the one case, it was probably tubercular; in others, it apparently proceeded from collapse and simple productive inflammation. The left side was affected in two females and in one male, and the right in two females. In all of them, the heart was more or less displaced, according to the extent of the process; especially so in the following:—

Miss A. K., aged twenty-nine, presented herself on the 7th of July, 1883. She complained, that she had suffered from Asthma since her third year. The attacks had always passed off within a few days, and had left her breathing tolerably free. For the last ten days, however, the dyspnœa had been persistent, so that she had been unable to lie down. Her cough had been very troublesome. The sputum was said to be thick, yellow and very tenacious. She had complete anorexia and diarrhœa. She also mentioned that she had shivering fits in the mornings, so that she was quite blue "with cold"; but that, towards the afternoon, she became burning hot.

The following history was given. Her mother was a confirmed invalid, and two brothers had always been delicate. She herself had had whooping cough at the age of three, and since then, she had been asthmatic. She had, moreover, been subject to sneezing fits and sore throats as long as she could remember. At the age of thirteen, her tonsils were cut. Her attacks returned once every six weeks or two months, and generally lasted from three to five days; copious micturition generally indicated their approach. She was always worse in the summer, than in the winter. She had never raised blood, and the sputa at the end of the paroxysms were scanty, greyish white, thick and tenacious. Her appetite was bad, and she had much discomfort after food, but her bowels acted regularly. Her catamenia began at eighteen years, returned every five or six weeks, and the loss was generally slight.

Patient was of small stature, of child-like proportions and of rickety conformation. She was very anæmic and emaciated, and had thick projecting lips. Temperature at 12 o'clock noon, 99° F. Pulse 96, very small and soft. Both nostrils were obstructed, and there was granular pharyngitis. There was slight kyphosis, the cervical glands were enlarged, and the fingers of the right hand clubbed. The thorax was small, deformed, and the ribs were angular. R. 24. The right side lagged behind, but there was no retraction. Here, in front, from the apex to the fourth rib, the resonance was tympanitic, and the breathing cavernous, accompanied by bronchophony. From the fourth rib downwards, the dulness was absolute. In the fourth right interspace, inside the nipple line, the cardiac impulse could be distinctly seen and felt. There was distinct superficial grazing, and the cardiac sounds were loud and clear. From the right supra-spinous fossa to the angle of the scapula, there were dulness and bronchial respiration; and thence towards the bases, the resonance was tympanitic and the breathing feeble. The left side was in front hyper-resonant, the respiration loud at the apex, feeble at the base, and accompanied by mucous rhonchi. The cardiac sounds were feebly conducted towards the normal area. Behind, and on the left, the resonance was impaired and the breathing was loud, and there was abundance of rhonchi.

On subsequent examination, the sputum was found to contain fibrinous cords, and the specimen of urine was not abnormal.

Broncho-pneumonia.

The clinical phenomena of the acute form of Asthma—the initial rigor, the high temperature, the pain in the chest, the great dyspnœa, the various kinds of stenotic sounds, and a more or less extensive dulness—may readily lead to the supposition, that the alveolar portion of the lungs participates in the inflammatory process of the bronchi. But the dulness is, as a rule, so mobile, so rapidly changing from one place to another, that it can be due only to congestion and atelectasis, and, in no wise, to infiltration and exudation.

Still, in many cases, a broncho-pneumonia apparently complicates Asthma, although the diagnosis of that affection is here as uncertain as it usually is. Its onset may, however, be suspected, if the dyspnœa loses its paroxysmal character and becomes continuous; if the temperature assumes a somewhat remittent type; if the physical signs remain stationary at some, generally the basal, portions of the chest; and if the fibrinous, are replaced by mucous or muco-purulent sputa, which are not infrequently streaked with blood, and contain, at the same time, a great abundance of large and swollen alveolar epithelium. In children, there is often a distinct flush on the side of the face corresponding to that of the seat of the pulmonary inflammation. In these circumstances, the chest clears up only gradually or imperfectly; and recovery from the attack may thus be considerably delayed.

The symptoms are much more distinctive if the broncho-pneumonia occurs in the interval between the attacks. In the midst of what they consider perfect health, adult asthmatics are seized with intense pain in one side of the chest, and, as a rule, also with rigors. The temperature rapidly rises to 104° F. or higher; respiration is more than thirty; the pulse exceeds one hundred, and is always of diminished tension and low wave. The sputa are copious, viscid, muco-purulent, and occasionally streaked with blood, and contain the finest fibrinous cords. There is loud tympanitic resonance on the painful side in front; behind, generally towards the base, percussion is dull, if the process is extensive, and the respiratory sounds are masked by various kinds of catarrhal rhonchi, which are heard also above and on the other side. Such state may last with unabated severity for two or three weeks, but generally terminates in recovery.

Croupous Pneumonia.

A typical attack of croupous pneumonia was observed in one man, aged sixty-five years, who had been asthmatic from infancy. The disease terminated fatally in eleven days.

In another case, that of a man aged forty-five years, who had also been asthmatic from birth, it was protracted for six weeks, but ended in complete recovery.

Rapid Phthisis.

Rapid phthisis was observed in one case of a female, in whom it was probably induced by the puerperal state and by lactation.

Mrs. C., aged forty-five years, presented herself in June 1881, and complained of dyspnœa, which was increased on the slightest exertion; of troublesome cough attended by difficult and frothy expectoration; and of great irritation of the nose and throat—irritation from which she had suffered as long as she could remember at that time of the year.

Patient had been subject to Asthma from infancy. She was of middle stature, of rickety conformation, with deformity of the chest and slight kyphosis; she was very anæmic and badly nourished. She had had two children; one of them was ten years old, and she nursed the other, four months old.

On examination, it was found that she had marked dulness, with great resistance of the left side, in front from the apex to the fourth rib, behind from the supra-spinous fossa to nearly the angle of the scapula. Over the dull part, there were bronchial respiration and bronchophony; while sonorous and sibilant rhonchi were heard at the left base.

The right supra- and infra-clavicular, and a space of about one inch and a half square in the right midscapular, regions, had impaired resonance and were the seat of rhonchi. Resp. 24. Pulse 72, very small and soft. Cardiac impulse was feeble, and inside mamillary line; the sounds were clear, but feeble. Temp. 101° F. She was advised to wean her baby, but she protested that the nursing did her no harm, and went on with it.

She was seen again in October. There had been repeated attacks of hæmoptysis, and signs of softening were observed on the left side. In December, there was unmistakable evidence of excavation. Night sweats and rapid emanation had set in. In March, 1882, the same symptoms persisted, and shortly afterwards, she died.

DISEASES OF THE HEART.

Hypertrophy and Dilatation of the Right Ventricle.

Hypertrophy, and probably also dilatation, of the right ventricle are an almost constant occurrence in Asthma. They arise from the increase of pressure in the pulmonary artery, either in consequence of an imperfect development of the lungs, or of the destruction of the capillaries in emphysema.

Sooner or later, there appear signs of cardiac weakness, as shown by a soft and small pulse, and by an amount of cyanosis which is altogether disproportionate to the extent of the bronchial obstruction. Anasarka is rare, probably because, with the general marasmus, there is a corresponding diminution of the whole mass of blood. It was observed only in three cases, in which the nutrition was fairly maintained. In two of them, however, there were varicosities of the lower extremities.

Valvular Lesions.

Only in one, out of one hundred and eighty cases, was there evidence of an organic lesion of the mitral valve. The patient was a young man, seventeen years old, who had been asthmatic from infancy. He was of middle stature, very anæmic, badly nourished and slightly cyanotic. He had marked kyphosis, and a peculiar deformity of the base of the thorax. The cardiac impulse was forcible in the fifth interspace, rather diffuse, and within the mamillary line. The cardiac area was enlarged towards the right. There was a loud systolic bruit at the apex, and the second pulmonary sound was distinctly accentuated.

Functional Derangements.

Over all the orifices of the heart, a soft blowing systolic bruit was heard in two females, each thirty-five years old, chlorotic, and emphysematous, who had suffered, for many years, from hay-fever, and had been asthmatic for twenty years. There was no secondary hypertrophy, nor dilatation, nor accentuated second pulmonary sound. Both alleged that they had had attacks of rheumatism.

A persistent slowness of the pulse was noticed in four cases—three males and one female; and, although there were several suspicious signs of chronic myocarditis, they were not sufficient to establish the diagnosis.

Mr. W. S., aged twenty-two years, asthmatic from infancy; very tall, with thoracic deformity and marked emphysema, had a pulse which, during a prolonged observation of several months, never exceeded fifty-four per minute, but generally ranged between thirty-eight and forty-two. Besides great cyanosis during the attacks, there were no signs of cardiac insufficiency.

Mr. E. S., aged fifty-seven years, subject to Asthma for twenty-five years. Cyanosis; small stature; deformed and small chest; pulse between forty and forty-eight; large liver, no icterus; slight anasarka of the lower extremities.

Mr. B., aged sixty-two, subject to Asthma for eighteen years, very tall, broad but flat chest, slight cyanosis, considerable anasarka extending beyond the knee-joints. Pulse persistently between thirty-eight and forty.

Mrs. L., aged forty, subject to Asthma from infancy, very tall, with pale and tumid face, fairly nourished, deformity of chest, had frequent fainting fits. Pulse always between forty and forty-eight.

Disease of the Vessels.

Sclerosis of the arteries is frequently seen in the comparatively young. In one man, aged forty, there was marked arcus senilis. Varicosities of lower extremities were seen in nearly 7 per cent. of all cases.

DISTURBANCE OF THE DIGESTIVE ORGANS.

Persistent icterus, apparently connected with intestinal catarrh, occurred in two females and in one male. The liver was not enlarged, but was painful on pressure.

Hæmorrhoidal flux is frequent, and the loss often very considerable, especially during the paroxysms.

Intestinal worms—*Ascaris lumbricoides*—were seen in two cases: in a child eight years old, and in an adult forty-two years of age. Their discharge had no influence whatever upon the return of the dyspnœa.[1]

DISEASES OF THE KIDNEYS.

The following five cases presented symptoms of renal disease:—

Mr. J., aged fifty-four years, had pain across the loins and hæmaturia during, and for some time after, the paroxysms. Gravel or stone was never passed, nor was there ever an attack of renal colic.

Mr. C., aged sixty years, had seven years before the first attack of Asthma all the characteristic signs of granular atrophy. He had had one attack of gout in the big toe, and had repeatedly passed small uric acid calculi. Within the last five years his Asthma had more frequently returned ; a few weeks before death, which took place in the country, there were no signs of uræmia.

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[1] Bergson, *Op. cit.*, p. 113.

The third case was remarkable for a persistence of pain which was seated in the loins, and extended thence to the scrotum and to the thighs. The pain was generally increased by the recurrence of the dyspnœal paroxysm.

Mr. G. T., aged thirty-nine, stated that, with the exception of great liability to "colds," he had enjoyed good health till 1879. He had then had an attack of pneumonia, on account of which he had been laid up for several weeks, and ever since, his breathing had been oppressed on the slightest exertion. At night he had frequently had the most painful attacks of orthopnœa. He had, for a long time, been unable to lie down, but of late, he had found some relief from inhaling the fumes of burning nitre paper. At the same time, he complained of pains in the loins, and added that they became considerably aggravated on the return of a dyspnœal seizure. He had also, in the winter of 1880, an attack of gout in the right foot, then one in the left big toe, and latterly one in the left knee. His appetite was bad, his bowels were regular, and he had not strength enough to attend to his work, as an engineer. He had also lost a great deal of flesh.

Stat. Pres.: June 14th, 1881.—Middle stature. Great pallor. Œdema of the face. Slender bones and thin muscles. Obstruction of left nostril. Enlarged tonsils. Chronic pharyngitis. Narrow and flat thorax. Lordosis. R. 24, costoabdominal. Expansion equal on both sides. Dulness and bronchial breathing in left supra- and infra-clavicular regions, and from left supra-spinous fossa to the angle of the scapula. Left base behind, tympanitic resonance and sonorous rhonchi. Right front and back hyper-resonant, feeble respiration, and distant rhonchus on cough. Sputa about four ounces in twenty-four hours, of greyish colour, viscid, containing large fibrinous cords. Cardiac impulse not seen nor felt. Cardiac area in fourth interspace from left parasternal to maxillary lines. P. 48, regular, very small and soft. Temp. subnormal. Tongue coated. Anorexia. Bowels open. Pain on pressure of abdomen and in lumbar region. Urine in twenty-four hours, about forty ounces. Sp. Gr. 1010, of a pale and yellow colour, acid, $\frac{1}{12}$ vol. albumen. Small hyaline casts.

Between June 14th and July 12th the dyspnœa somewhat abated. There were signs of chronic uræmia, as headache, giddiness, slight somnolence, which, however, subsided on the spontaneous occurrence of from ten to twelve watery discharges from the bowels. The quantity of the urine increased to 60 oz., and the Sp. Gr. to 1015. P. 60, gaining a little in force. Increase in weight of about 4 lbs. Appetite improved, and the pain in the back also somewhat diminished. The patient was then lost sight of.

In one female, Mrs. C.,[1] there was amyloid degeneration of the kidneys, liver and spleen, in consequence of collapse-induration of the right lung, general emphysema with bronchiectasis, and considerable hypertrophy of left ventricle.

In the fifth case, hæmorrhagic nephritis supervened upon one-sided shrinking of the lung.

Miss D., æt. 30, whose father had died of phthisis, had since her seventh year of age been subject to hay fever. At sixteen, she had had Asthma, in consequence of which she had been laid up for several weeks. Since then the Asthma had returned every summer. Latterly she had had a great deal of dyspnœa, cough, and repeated attacks of hæmoptysis.

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[1] *Vide* p. 75.

Stat. Pres.: June 1883.—Tall; face slightly œdematous and cyanotic. Considerable emaciation. R. 24. P. 96, small and soft. Nose pervious. Chronic pharyngitis. Thorax small and flat. Imperfect expansion of left side. Absolute dulness, increased resistance and bronchial breathing, left apex front. Left supra-spinous fossa dull. Left base behind tympanitic resonance, mucous rhonchi. Right apex front, tympanitic resonance and loud respiration. Cardiac impulse rather forcible, diffuse between second and fourth intercostal spaces; apex beat near anterior axillary line. Sounds loud and clear. Sputa muco-purulent, fibrinous cords. In the autumn of 1887 she was reported to have been suddenly seized with a painful swelling of the left wrist-joint, which had lasted for some time and had been followed by rebellious diarrhœa, anorexia, and vomiting. Recovery. She was again seen July 1, 1888. There was great anæmia and marasmus. Œdema of the face and extremities. Very small and soft pulse. Dry and coated tongue. Complete anorexia. Urine rather copious, of dark brown colour, acid, nearly ½ vol. albumen. Shortly afterwards Exitus lethalis.

Gout and Rheumatism.

In four of the cases previously mentioned, there were painful swellings of the joints. In two of them, the articular affection was, probably, of gouty origin; in one of them it completely subsided on the appearance of the Asthma, in the other it persisted simultaneously with the dyspnœa. In the remaining cases, there was no evidence of a gouty diathesis, but in all of them there were symptoms of renal disease.

DISEASES OF THE SKIN.

Various forms of skin diseases were observed in nearly one-fourth of all the cases. As a rule, they manifested themselves simultaneously with the first symptoms of Asthma. They are chiefly prevalent in the young, and the tendency to them persists so long as the tissues of the cutis retain a certain degree of softness. With advancing age, they generally subside, and only in one case was eczema of the hands observed in a man, aged sixty-two years, who had, at frequent intervals, suffered from it for forty years.

In some instances, every acute attack of Asthma is accompanied either by a few herpes vesicles of the lips, or by a diffuse eruption of them all over the face. In others —in five out of one hundred and eighty cases—papular efflorescences, which cause some itching, appear on the chin or on one side of the neck ; they are of a dark blue colour, are not converted into wheals or vesicles or excoriations by scratching, but soon disappear without leaving any desquamation of the epithelium or pigmentation. Hyde Salter is inclined to think that the "itching under the chin" is universally present, or, at all events, most strongly marked, in the Asthma that accompanies hay fever.[1] Occasionally, erythema of the fingers or of the whole body occurs with, or between the dyspnœal paroxysms. In one case, there was an attack of urticaria, and Phœbus also reports to have, once, observed the latter in connection with "hay fever."[1]

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[1] *Op. cit.*, p. 73.

The most frequent complication on the part of the skin is, undoubtedly, eczema. At times it is limited to the fingers or hands, but oftener it affects also the face, the back and the chest. A fresh outbreak, or a recrudescence of it, generally takes place with the return of the acute attack of Asthma; and while the eczema steadily passes through its various stages, the dyspnœal paroxysms considerably abate. Hence, the theory that "the disease" alternately implicates the skin and the lungs, the one organ improving under the disease of the other. If the researches of Gifford,[2] who attributes the vesicular eruptions to the presence of a streptococcus, are confirmed, it would appear that Asthma and eczema are co-ordinate symptoms.

DISTURBANCES OF THE NERVOUS SYSTEM.

Symptoms on the part of the nervous system are rare. Insomnia, however, at the time when the breathing is quite unimpeded and the skin free from all irritation, is frequently complained of, and is not peculiar to those who are accustomed to the use of narcotics.

Epilepsy existed in two cases. One of them was that of a man fifty years old, who had had Asthma for twenty years. The first attack of epilepsy came on six years ago, at the very moment that he consulted a medical man at Ems. The kidneys were not affected. His Asthma persisted in spite of the complication.

[1] *Op. cit.*, p. 31.

[2] *Arch. f. Augenheilk.*, Bd. 16, p. 197, quoted by Klebs, *Die Allgem. Pathologie*, 1887, p. 495.

The other was that of a man, fifty years old, who had been asthmatic from infancy. Epilepsy supervened within the last three years. Latterly he had also attacks of mania, and raved, during his dyspnœal paroxysms, as much as his breathing powers would permit him. On the advice of a specialist, he was removed to an asylum.

Paralysis agitans existed in one man sixty-six years of age, and asthmatic for thirty years. Another, sixty-two years old, and subject to Asthma for fifteen years, had hemi-atrophy of the left side of the face and of the left sterno-mastoid muscles, supposed to have developed itself in consequence of a fall.

Many asthmatics object to warm rooms, and even in the midst of winter prefer to sit without fires. Their temperatures were repeatedly taken in the intervals between their paroxysms, and were found to be subfebrile.

GENERAL NUTRITION.

Where the air passages remain occluded for any length of time, compensation is established by a reduction of the respiratory wants. The breathing becomes habitually shallow, and there is an increased loss of albuminates. Asthmatics, are, consequently, always in a state of considerable anæmia and emaciation. In the obese, the loss of weight is, under similar circumstances, less marked than the increase in the anæmia.

In three cases—two females and one male—an improvement of the Asthma was followed by a rapid development of an enormous obesity. Both females were at the climacteric period; the one was married and had had several children, the other was single. Both had an hereditary disposition to corpulence. So long as they were subject to the dyspnœal paroxysms, they were very thin; but on their recovery, the catamenial losses became very copious, and, in an incredibly short time afterwards, an abundant deposition of fat took place all over the body.

In the one male, the corpulence was preceded by a repeated and copious hæmorrhoidal flux.

COINCIDENCE WITH ACUTE DISEASES.

In three children—two of them under ten years of age, and one twelve years old—who had been asthmatic from infancy, attacks of measles and whooping cough were observed. Neither of these affections was unusually protracted nor otherwise severe.

In one of them, a girl eight years old, who had considerable kypho-scoliosis, the measles had apparently a beneficial effect upon the asthmatic paroxysms. In the course of the disease, the fibrinous sputa became muco-purulent; the expectoration was comparatively easy, and helped to clear up the chest. For some time afterwards, she was freer from dyspnœa than she had ever been before. Such improvement is readily intelligible from the difference of the pathological processes; for, in the one case, there is a fibrinous exudation, which rapidly coagulates into a firm and elastic material; in the other, a peptonising agency is at work, softening and liquefying the inflammatory products.

Similar amelioration of Asthma has been observed also in adults, in consequence of a bronchitis of septic origin.

CHAPTER VII.

DIAGNOSIS OF BRONCHIAL ASTHMA.

A. Diagnosis of the Disease: Significance of the Sputa; Indications from the History and the Present State.—B. Diagnosis of the Paroxysms—Differential Diagnosis: *a.* Inspiratory Dyspnœa; *b.* Cardiac Dyspnœa; *c.* Dyspnœa from Spasm and Paralysis of the Diaphragm; *d.* Dyspnœa from a Combination of Causes.

A. DIAGNOSIS OF THE DISEASE.

Significance of the Sputa.

THE diagnosis of Bronchial Asthma, at the time when the paroxysms have completely subsided, is greatly facilitated by the characteristic traces, which they have left behind them. In some instances, the patients continue to raise, especially in the morning,—even if their breathing is absolutely free—a few greyish, transparent pellets, which contain the hyaline cords previously described. From their presence the nature of the past dyspnœa may be inferred with a degree of probability, amounting almost to a certainty.

The diagnostic value of those peculiar formations is in no way impaired, as it has been urged,[1] by their reported occurrence in cases of pneumonia. Rightly interpreted, they indicate only a form of fibrinous bronchitis. But though their presence need not be, and indeed is often not, accompanied by dyspnœa, still the affection of which they are a symptom leads, as a rule, to a transient stenosis of the larger tubes. Hence, if they are met with in connection with attacks of Asthma, there can be little doubt, that they are concerned in the production of the functional derangement. That they have no such effect in cases of pneumonia is quite intelligible from the difference of the pathological conditions. In the earlier stages of that process, they are retained at their place of origin by the rigid alveolar exudation with which they are contiguous; later on, they participate in its softening or puriform melting, and thus lose their power of causing obstruction.

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[1] Vierordt. v. Jacksch and others; *vide* p. 27.

Nor does it detract from their significance that, occasionally, none are found in the sputa of typical cases of Asthma.[1] Their absence cannot safely be affirmed, unless after the application of a certain test. For, in the young and the feeble—in whom the cough is deficient in force—as also in complications with dilated bronchi, they are, in consequence of their prolonged retention in the air passages, uncoiled and altered in their appearance, so that they are readily concealed by the preponderating mass of mucus. Thus it happens, that a large number of microscopic preparations, spread as thinly as the tenacity of the material will permit, may be examined without detecting any of them; on the addition, however, of a drop of a solution of caustic potash, they are at once rendered visible.

Indications from the History and the Present State.

Frequently, there are no sputa in the intervals between the paroxysms. The exudation has either already been expectorated; or what remains of it is too firmly impacted for removal by the habitually shallow, and even by the deliberately forcible, respirations of the patients. The diagnosis has then entirely to rely on the analysis of the history and of the actual state of the case. A careful scrutiny of the available data may thus lead to a presumption in favour of the disease—a presumption which becomes the stronger, the more numerous are the circumstances which point in the same direction. At times, however, the facts are so scanty, and, indeed, so ambiguous, that no reliable clue can be obtained, all efforts notwithstanding, as to the nature of the previous dyspnœa.

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[1] *Pel. Zeitsch. für klin. Mediz.*, Bd. ix., Heft 1.

The history of the case affords some indication. Most forms of paroxysmal dyspnœa suddenly arise in the midst of seemingly perfect health; and often, by their appearance alone, attention is first directed to serious organic lesions, which must have existed for some time. Bronchial Asthma, on the contrary, invariably gives fair warning of its approach. Almost each attack has, as already mentioned, its premonitory symptoms. The disease itself assumes its characteristic features only after prolonged ailments. For an indefinite period, its development is preceded by inflammatory irritation of the respiratory mucous membrane. It is generally alleged to be traceable to pneumonia, to congestion of the lungs, and to bronchitis; moreover, there is generally an admitted liability to "colds," or, a disposition to the so-called "hay fever." The earnest assurance of some patients that, before they became asthmatic, they "never had a day's illness," must be cautiously received. On careful inquiry into this matter, it will be found that coughing and sneezing, however troublesome these symptoms may have been, are yet comprised within the limits of health.

Of equal diagnostic importance is the time of life, at which the disease first manifested itself. Bronchial Asthma most frequently develops itself in infancy and childhood; but with the advance of age, the tendency to it considerably diminishes, without ceasing altogether. The presumption is, therefore, against it, if, in otherwise doubtful cases, the recurrent form of dyspnœa suddenly commenced at or after the fortieth year. It is needless, however, to add that in children also the paroxysms may have proceeded from various causes. Amongst these may be, for instance, the so-called pseudo-croup in connection with chronic tonsillitis. Many an access of it is erroneously reported as Asthma. The absence of deformities of the chest, and a fair state of the general nutrition, will, however, help towards a decision on its nature.

In a number of cases, percussion and auscultation give negative results; and there is, thus, no positive evidence of pathological changes in the lungs and the heart. True, the absence of appreciable signs does not prove that those organs are sound, especially not in an individual, who is subject to attacks of Asthma. Indeed, a suspicion to the contrary is roused by the entire habit which the patient invariably presents—by his whole configuration, by his profound anæmia, and often by his marasmus. But the difficulty is that, from these alone, no safe inference can be drawn as to the nature of the occasional obstacle to respiration. In the emaciated, undersized, or overgrown, with the conspicuous deformities of the whole osseous system; and in the anæmic obese with the rickety conformation of head and limbs, it may be surmised, for reasons previously stated, that the past disturbance proceeded from a transient stenosis of the bronchi. Such diagnosis, however, would be nothing but mere conjecture; for this constitutional defect may have led to derangements of various kinds, from all of which the dyspnœa may have arisen.

In the great majority of them, however, organic lesions, varying in kind and extent, are plainly discernible. Some of them, judging from their rare occurrence, appear to be only accidental complications. Others, on the contrary, are so frequently observed in the circumstances, that they must be either remote causes or consequences. Still, whatever they are, they are manifestly consistent with a certain freedom of respiration, and allow, therefore, no immediate inference as to the nature of the past disturbance.

It is known, however, that certain pathological conditions are more frequently associated with one form of dyspnœa than with another. Upon that ground, catarrh of the air passages, or rather the various stenotic sounds, and emphysema, would be suggestive of Bronchial Asthma, of which they are an almost integral part. Valvular lesions of the heart, or degenerative changes of its muscles, would point, for a similar reason, to cardiac dyspnœa. But it does not follow that it must be so. Neither the emphysema nor the cardiac affection excludes other forms of dyspnœa. The difficulty is even greater, if there are, at the same time, morbid changes in several organs, each of which may have in various ways caused the asthmatic paroxysms. Nor would it be admissible to assume a definite form of Asthma—the so-called uræmic,—from the mere presence of diseases of the kidneys; for even if the dyspnœa could be convincingly traced to them, it may have arisen either from œdema of the glottis, from cardiac insufficiency, or from fibrinous bronchitis.

But the diagnosis may, with greater probability, be established, if the physical signs point to a bronchial stenosis. In such cases, there are either over one lobe—the lower oftener than the upper—or over an entire lung, complete suppression of all breath sounds; an equal expansion on both sides during shallow respiration; either normal or but slightly impaired resonance on percussion; and imperceptible vocal fremitus. Repeated acts of coughing may restore the perviousness of the air passages, and give rise to sonorous and sibilant rhonchi. Often, however, they have no influence upon the obstruction, and the silence persists. If the history of the case and the associated phenomena exclude the entrance of a foreign body from without, a dilatation of the bronchi or a neoplasm, the obstacle most likely proceeds from a fibrinous exudation, or from the products of its regressive metamorphosis; for neither the catarrhal nor the puriform secretion has sufficient cohesion to produce those symptoms, so long as the patient is conscious.

B. DIAGNOSIS OF THE PAROXYSMS.

In the presence of the paroxysms, Bronchial Asthma can readily be recognised, even if the history of the case be unknown. Indeed, the physical signs are so characteristic as to indicate at once the nature of the dyspnœa. The diminished frequency of respiration; the partial distension of the thorax, with the retraction of its apex and its base; the hyper-resonance of some parts and the dulness of others; the short and forcible inspiration, and the persistence of the inspiratory position, notwithstanding the active and prolonged expiration; the continual changes between suppression of sounds and the sibilant and the sonorous rhonchi;—all these conclusively point to a stenosis of the larger tubes by a movable pathological product of great tenacity. In private practice, the demonstration of the actual obstacle is at the time impracticable; and, unless it were to confirm the diagnosis, the subsequent microscopic examination of the sputum would almost seem superfluous; for such a series of symptoms can only proceed from a fibrinous exudation into the bronchi. A prolonged and imperfect expiration might perhaps arise from a pedunculated tumour in the subglottic region; but, apart from the rare occurrence of such a tumour, inspiration would be easy, and the above phenomena on the part of the chest would be absent.

The physical signs, peculiar to Bronchial Asthma, may, however, to some extent be modified by various complications, of which insufficiency of the cardiac muscles is the most common. As this considerably increases the frequency, and reduces the depth of respiration, the distension of the thorax is consequently less marked, and the pitch of the rhonchi is altered. If, at the same time, there are cyanosis and œdema of the ankles, or adventitious sounds at the heart with or without enlargement of its area, the question may arise, whether the dyspnœa may not be due to disease of the organ of circulation and to secondary catarrh.

So also in hydræmia, especially in connection with kidney mischief, a *hydrops ex vacuo* may readily develop itself in the partially occluded tubes. The abundance of the moist sounds, the copious and frothy expectoration of saliva, the occasional suction of the latter into the windpipe and the consequent tracheal rattle, are apt to suggest an œdema of the lungs. But though, in either of these cases, the complications have some share in the production of the dyspnœa, yet its main cause is clearly shown by the prevalence of the expiratory stenosis.

DIFFERENTIAL DIAGNOSIS.

All other forms of paroxysmal dyspnœa have nothing in common with Bronchial Asthma except the painful effort in supplying the respiratory wants. They differ from it in their modes of origin, and accordingly also in their clinical manifestations.

(*a*) *Inspiratory Dyspnœa.*

Thus, one group of them is distinguished by prolonged and forcible inspiration, less difficult or even normal expiration, loud stridor audible at a distance, imperfect expansion of the thorax, and a retraction of its apex and base. It comprises all cases, in which an obstacle is situated at or above the bifurcation of the trachea, impeding the access of air to the alveolar surface. In most of them, the nature and the exact site of the stenosis are readily accessible to diagnosis. In some, there are pathological changes in the larynx, which are seen at once on laryngoscopic examination. In others, there is evidence of either compression or constriction of the windpipe.

If laryngeal inspection is impracticable, or if no tracheal compression is visible, the stenosis may be localised where the stridor has its maximum intensity. Seated in the larynx, it is, probably, due :—to œdema of the glottis, if it occurred during an inflammatory affection of that part, or in connection with chronic Bright's disease; to paralysis of the posterior crico-arytenoid muscles, if the present attack is merely an acute exacerbation of a chronic and afebrile form of inspiratory dyspnœa, not accompanied by alteration of the voice, or if there are symptoms of hysteria; to a tumour, in case of chronic hoarseness; or to a foreign body, if, especially in children, its onset was sudden, in the midst of perfect health, and under otherwise suspicious conditions.

The nature of the tracheal stenosis is, in the circumstances, much more difficult to determine. Still if, from the history of the case, constitutional syphilis, and consequently cicatricial shrinking of the mucous membrane, can be excluded, the possibility remains that a tumour or an aneurism may, as yet, be so concealed as not to betray its presence.

A foreign body in the windpipe or in a main bronchus may be suspected upon the same grounds as it is in the larynx. Occasionally, especially if its entrance was unperceived, it is apt to simulate Bronchial Asthma, if its shape and size are such, that on inspiration it is sucked towards the periphery, and on expiration, propelled towards the centre, but somewhere arrested. The change of the objective signs from one side of the chest to the other is not so pathognomonic as is generally supposed. A decision, however, may be arrived at, on due consideration of the origin of the disease, and, on an examination of the sputum.

The croupous inflammation of the trachea or of the main bronchi—both in its idiopathic and in its secondary forms—presents the inspiratory type so long as the exuded material is a hollow cylindre and firmly adherent to its matrix. Its existence may, with great probability, be surmised from the intensity of the dyspnœa, which suddenly develops itself in the course of what seems to be a severe catarrhal affection. The diagnosis is generally confirmed by the characteristic sputum, provided the large white or greyish masses, which not infrequently are coated with blood or mucus, are at once unfolded in water. But if the exudation is only partially detached, so that a portion of it becomes floating, all differences cease between the anatomically definite croupous bronchitis and the clinically definite Bronchial Asthma, with which it is, indeed, identical in its etiology, its pathogeny, and its manifestation.

Spasm of the glottis—the so-called Asthma thymicum or Asthma Millari—bears even less resemblance to Bronchial Asthma than any of the forms of dyspnœa hitherto mentioned. It generally occurs in infants of a rickety constitution, although the same symptoms may occasionally be observed also in adults, in whom they probably proceed from a different cause. In either case, the laryngeal obstruction is so considerable that respiration is momentarily altogether arrested, there is loss of consciousness, and in children convulsions take place.

(*b*) *Cardiac Dyspnœa.*

In another group, the forcible respiratory movements are rhythmical and very frequent; the expansion of the chest is uniform; the air passages are pervious, and there is evidence of cardiac insufficiency. In view of the ample supply and the free circulation of the air, the sudden dyspnœa can be due only to the blood not being carried in sufficient quantity and at regular intervals to the surface of the lungs. Those symptoms represent the Cardiac Asthma of some writers, and occur in the cases, in which the contractile energy of the ventricles temporarily fails, or in which a main branch of the pulmonary artery, or a corresponding number of its radicles, is temporarily obstructed.

The failure of the cardiac muscles may be assumed if the series of morbid phenomena just referred to, is observed in the later stages of hypertrophy and dilatation of the heart, either in its idiopathic form, or in that, secondary to valvular lesions, to sclerosis of the arteries, to Bright's disease, or in Diabetes. It is said to be produced also by gummata of the heart,[1] which, however, are not accessible to a clinical diagnosis.

Embolism of the pulmonary artery presents no characteristic signs by which it could be recognised with any degree of certainty. It can be merely suspected, if a source of it is known to exist, and if, at the same time, the resonance over a whole lung, or over its greater part, is normal, and the respiration harsh; whereas over the rest of the chest, there are symptoms suggestive of a collateral œdema.

Œdema of the lungs, which is not of a collateral origin, occurs most frequently as a complication with chronic diseases of the heart and the kidneys. The copious serous exudation impairs to some extent the resonance of the chest, and gives rise to all kinds of moist sounds. There is also an abundant and frothy expectoration of serum, mixed with blood.

(*c*) *Dyspnœa from Spasm and Paralysis of the Diaphragm.*

Again, there are cases, in which the forcible movements are limited to only one set of the respiratory muscles—those of the upper part of the thorax,—while, on examination, no impediment can be found in the air-passages, and none inherent in the pulmonary circulation. Those movements are therefore intended either to compensate for a defect, or to oppose an excess, in the contractile energy of the diaphragm.

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[1] G. Rosenfeld, *Syphilitisches Asthma, Med. Corresp. Blatt d. Würtemb. Aerzte Ver.*, No. 38, 1882.

In the tonic spasm of the diaphragm,—an affection of rare occurrence—the vertical diameter of the chest is increased, the base is distended and fixed, the epigastrium is projecting, and there is active expiration.

In the paralysis of it—equally rare—the base is not expanded during inspiration, and the epigastrium is, on the contrary, retracted.

The physical signs are similar in a mechanical interference with the action of the diaphragm, such as is said to arise from extreme tympanitis. The individuals, however, subject to this form of dyspnœa generally present symptoms of cardiac insufficiency, which not improbably has a considerable share in the production of the disturbance.

(*d*) *Dyspnœa from a Combination of Causes.*

Not infrequently it happens that a paroxysm proceeds from a combination of several pathological processes. In such cases, an analysis of the manner in which the mechanism of respiration is deranged, will serve to establish the diagnosis.

CHAPTER VIII.

COURSE AND PROGNOSIS OF BRONCHIAL ASTHMA.

Course of the Acute and the Chronic Forms—Prognosis of the Paroxysms—Prognosis of the Disease—Mortality—Effects of Treatment.

Course of the Acute Form.

Bronchial Asthma, in its acute form, runs a typical course, passing through the successive stages of inflammatory hyperæmia, of exudation, and of a transient stenosis of the main tubes. The differences, which it presents, are only such as arise from the varying degrees in the intensity and the duration of the pathological process.

Occasionally, all the symptoms may subside at the end of a few days. During that time, the dyspnœa makes but short and imperfect remissions, owing to the persistent obstruction of the air passages. The continuance of the forcible respiratory movements has, however, a beneficial effect: it prevents the accumulation of the pathological products, the removal of which thus proceeds, as nearly as possible, *pari passu*, with their formation.

Often the disease is protracted for several weeks. The dyspnœa then assumes a more distinctly paroxysmal character, returning at intervals, during which everything seemingly tends to recovery. Its recurrence must be fully expected, so long as there are signs of a partial or complete imperviousness in the middle or lower portions of the lungs. Frequently, however, it takes place as a surprise not only to the patient, but also to the observer, who, on a previous and most careful examination, could detect no abnormality whatever. In that case, it is due, either to the detachment of an old plug, which lies in a bronchus, surrounded by healthy or emphysematous tissue, at some distance from the surface of the chest, respiration being too shallow to produce stenotic sounds; or, it is due to a fresh access of the exudation, as shown by a rise of temperature, and by a return of the inflammatory symptoms on the part of the nasal and pharyngeal mucous membranes. But there are no means of deciding, whether the process has relapsed at the place first affected, or whether it has invaded another, somewhat in the manner of the migratory form of pneumonia.

The recovery from those attacks is generally complete. The disturbances which they produce entirely disappear. The patients regain the breathing power, which they possessed before; and in the absence of complications, all other functions are performed, not perceptibly different from what they are in the healthy.

COURSE OF THE CHRONIC FORM.

In numerous instances, the disease uninterruptedly lasts for years, and the dyspnœal paroxysms return at shorter or longer intervals, often even once or several times in twenty-four hours. Possibly the process of exudation is in them, on account of complications, continuous, and the removal of the product inadequate. Still, from the marked improvement which may be, even here, effected, it would appear, as if such essentially chronic course were not an inherent peculiarity of the disease, but were to a great extent preventible.

PROGNOSIS OF THE PAROXYSMS.

Formidable as a severe paroxysm invariably is to both patient and bystander, it generally terminates favourably. Even such threatening symptoms as slight somnolence, extreme cyanosis, foam at the mouth, tracheal rattling, cold extremities, and absence of a pulse at the wrists, may completely subside. Nevertheless, a considerable degree of cyanosis during an attack always indicates a failing action of the heart, either from atrophous, or from myocarditic, changes in its tissues; and cases of that kind are not free from danger.

So far as could be ascertained, death took place, during the paroxysms, in five out of one hundred and eighty cases —*i.e.*, in nearly 3 per cent. But that number does not represent the exact state of the mortality during the attacks; for information was not obtainable as to the manner, in which the end came, in all that proved fatal.

Amongst those five were three of females and two of males. Only one of the former was seen a few hours before death.

1. Mrs. C., aged thirty-seven years, whose case has already been related, was seized with dyspnœa at ten o'clock p.m., and died at one o'clock a.m. The cause of death was plugging of the larger bronchi and chronic myocarditis.

The others died in the country or abroad, under circumstances which were reported as follows :—

2. Mrs. P., aged fifty-nine, subject to typical Asthma for more than twenty-five years, died within two hours after the commencement of what was supposed to be one of her habitual paroxysms.

3. Miss C., aged forty-nine years, and asthmatic for thirty, was under observation for a considerable time. Previously, she had had attacks almost every night, and almost every remedy had, in vain, been tried for their relief. When she presented herself, there were unmistakable signs of bronchiectasis and cardiac weakness. She soon, however, sufficiently recovered, to be able again to ride on horseback; and so long as she did not over-exert herself, she was tolerably free from dyspnœa. From August 1885 to the end of March 1886 she had no Asthma. She only complained of obesity, which had rapidly developed itself.

Shortly before her death, whilst in the enjoyment of otherwise good health, she underwent, in the country, two successful operations for the removal of a polypus, which had gradually formed in consequence of a chronic hyperplastic catarrh of the nose. On March 21st, 1886, she was, towards the evening, seized with, what she thought, the premonitory symptoms of an attack, during which she died two days afterwards.

4. Mr. C., aged sixty-nine years, had had, during the last ten years, five or six attacks of Asthma. There were symptoms also of granular atrophy of the kidneys and of chronic myocarditis. One of his paroxysms, lasting eight hours, proved fatal.

5. Mr. S., æt. fifty-six, was seen on February 2nd,

1882. He had been subject from infancy to "colds," and especially to severe fits of sneezing. Twelve years ago, he had had the first attack of Asthma, but he had previously had a great deal of wheezing in his larynx. His dyspnœa had generally been preceded by a feeling of constriction in the throat and by severe coryza. His paroxysms had lasted each time about three days, but they had frequently returned. In the intervals between them, his respiration had been free. His appetite had been fair, but he had had discomfort after meals. His bowels had been regular, and there had been some irritation at the anus.

Mr. S. was over six feet tall, 12 st. 6 lb. in weight, fairly muscular, and of sallow complexion. He had a very small head, a perfectly straight spine, and a comparatively small and flat thorax. His nose was pervious, the tongue coated with a thick white fur, which was almost contiguous with a similar substance on the corresponding portion of the pharynx. The tonsils were atrophied, and the mucous membrane of the throat was red and swollen. Respiration eighteen in the minute. There was marked hyper-resonance on percussion in front, extending on the right to the seventh, and on the left to the sixth ribs. Both lungs descended, on deep inspiration, nearly one intercostal space. Respiration was feeble above and at the base; but in both mammary regions sonorous rhonchi were heard after coughing. Behind, the resonance was considerably impaired on both sides, and outside the angle of the right scapula, there was distinct dulness. Over the latter place, there were bronchial respiration and bronchophony; over the rest, the respiration was feeble, and there was abundance of sub-mucous rhonchi from apex to base. Pulse was 72, regular, very small and soft. The cardiac impulse could neither be seen nor felt, and the cardiac area could not be made out. The sounds were feeble, but clear. The liver dulness was small; there was slight tympanitis, but no anasarka. The sputa contained the typical constituents of Bronchial Asthma, and the specimen of urine submitted for examination was of normal sp. gr., and free from albumen and casts. He was ordered Ammon. Benz., Camph. trit. āā gr.iij. t.d. In a fortnight's time he had considerably recovered. He was not seen again, but a friend of his whom he had, later on, sent to me, stated that he had remained quite free from dyspnœa.

Mr. S. died during an attack of Asthma, about nineteen months afterwards, at St. Moritz, and Dr. Holland was good enough to write to me as follows:

"On Tuesday, Sept. 23rd, he sent for me, and I found him in a dreadful state. P. 140, T. 104·4° F. There was an attack of Asthma as bad as ever he had had before, with acute bronchitis and the small tubes full of mucus. He was a great sufferer from rheumatism and gout, and was, when I saw him, taking thirty grains of salicin every two hours, which evidently had depressed the heart's action enormously.

"All I could do was of no avail, and he died asphyxiated just twenty-six hours after I had first seen him."

Prognosis of the Disease.

In the favourable circumstances, previously alluded to, the disease may remain for years in a state of abeyance. But it is doubtful, whether in any such case, its cessation may be looked upon as final; or whether the predisposition to it, which is often hereditary or congenital, is entirely eradicable. In the abstract, such contingency is not impossible; the facts, as they are daily met with, do not render it very probable.

No solid ground exists for the current belief that children almost always grow out of their Asthma. No doubt, there are adults who in their younger days have suffered from repeated attacks of dyspnœa, which subsequently have never returned. But the nature of that dyspnœa has not been ascertained; and no case, in which the diagnosis has been established, has as yet been observed for a sufficient length of time to admit, with certainty, the alleged permanence of the recovery. So far as facts go, there is evidence, on the contrary, that one-half of all the adult asthmatics have acquired their disease during childhood. In one hundred and eighty cases the ages at the time, at which the patients came under observation, were stated to be as follows:

AGES.	MALES.	FEMALES.	BOTH SEXES.
Under 10	8	6	14
10—20	20	7	27
20—30	14	19	33
30—40	13	15	28
40—50	18	16	34
50—60	13	12	25
60—70	14	4	18
70—81	1	...	1
Total	101	79	180

Deducting from that number the fourteen under ten years of age, whose prospects are at issue, and the eighty-two in whom, according to the table on p. 98, the disease commenced after the twentieth year, there remain eighty-four individuals of all ages, in forty-nine of whom it first appeared before the tenth, and in thirty-five between the tenth and the twentieth year.

It is difficult to absolve the current doctrine on the nature of the disease from all responsibility as regards the frequency of its recurrence. The theory is widely spread that Bronchial Asthma is essentially a spasm, the suppression of which is held to be the main object of treatment. Imbued with that idea, the patients become intolerant of the slightest restraint at the first sign of relief; and the reappearance of the dyspnœa is to them the surest proof that the adopted remedies have failed. Yet, in reality, the dyspnœa is a compensatory process, which, though exceedingly painful in itself, leads to a salutary end. It unmistakably shows the presence of an obstacle in the bronchi; and so long as the centre of respiration preserves its normal irritability, there can be no prospect of its cessation, till the pathological products have been removed.

That the prevalent views lead to serious practical consequences, is obvious enough. Although the organism, thanks to the auxiliary forces at its disposal, succeeds for a time in supplying its respiratory wants, notwithstanding an impediment in the air passages, still it cannot long and effectively do so under such conditions. If, therefore, a great part of the lungs continues to be excluded from its function, compensation can be established only by a reduction of the very wants themselves. Life, henceforth, is carried on, as it were, on a lower scale. The tissue-changes are perverted, and, instead of tending to the preservation of the body, they lead to degeneration of its component elements. The young and comparatively strong may, at first, resist the process; but ultimately they must yield. The obese not only maintain but increase in weight, doubtless at the expense of their albuminates, while all their organs become atrophic. The resistance is even less at a period of life, when the tendency to regressive metamorphosis is more or less normal. Hence, even if the appetite is unimpaired and there is no subjective dyspnœa, emaciation and anæmia become excessive, producing the typical aspect of chronic asthmatics.

It is evident, therefore, that the impairment of the general nutrition must necessarily increase the receptivity to the exciting cause. Nor is it improbable, that the accumulated exudation becomes, in its turn, a fresh centre of infection. That decomposing organic substances form a suitable nutrient medium for a virulent streptococcus is generally admitted; and there is no reason to suppose, that Asthma is, in that respect, exceptional.

The prognosis as to life chiefly depends upon the constitution of the patient and upon the course of the disease. If the paroxysms are of short duration and separated by long intervals of free respiration, the disturbances which they produce are readily repaired, age notwithstanding. Serious consequences result from them only, when they are unusually protracted, and when a considerable portion of the lungs remains obstructed. The general nutrition thus suffers, and, as just explained, the disposition to relapses is thereby increased. The power of resistance to any such inroads is necessarily greater in the young and strong, than in the old and decrepit, organism.

Complications with various organic lesions of the lungs, the heart, or the kidneys, render the prospects of life very doubtful. The accession of an acute disease, of a purulent form of bronchitis, or of a croupous pneumonia, is fraught with the greatest danger. It may here be mentioned, however, that in a large number of asthmatics, there are in the one or the other infra-clavicular region, absolute dulness with increased resistance, bronchial breathing and bronchophony; but all these signs, though otherwise suggestive of tuberculosis, as a rule, completely disappear within a few weeks.

MORTALITY.

The popular conception that Asthma does not perceptibly abbreviate the natural term of existence is probably founded on the fact, that some who are subject to it live up to, and over, seventy years of age. Those are undoubtedly exceptional instances, which do not prove the rule. Most of them have acquired the disease late in life, and their attacks run a typical course. Only two out of one hundred and eighty cases, in whom it developed itself in infancy, reached over sixty years; one of them was for twenty-five years in India, where he enjoyed an absolute immunity from it; the other came of a very long-lived family, and had, at first, only very short seizures and at long intervals.

Within a period of ten years, the mortality was at the rate of thirty out of one hundred and eighty cases, *i.e.*, nearly seventeen per cent. Amongst one hundred and one males, there was a death-rate of fourteen, *i.e.*, nearly fourteen per cent.; and amongst twenty-nine females, one of sixteen, *i.e.*, more than twenty per cent.

The ages at the time of death were as follows :—

AGES.	MALES.	FEMALES.	BOTH SEXES.
Under 20	1		1
20—30	1	1	2
30—40	—	3	3
40—50	2	4	6
50—60	1	8	9
60—70	7	—	7
70—80	2	—	2
Total	14	16	30

The youngest was nineteen, the oldest seventy-seven years of age. The greatest mortality of the females was between fifty and sixty; that of the males between sixty and seventy.

The duration of the disease in the fatal cases was ascertained to be the following :—

DURATION.	MALES.		FEMALES.		BOTH SEXES.
	AGES.	No.	AGES.	No.	
Under 5	„	„	34	1	1
5—15...	44. 57. 65. 69. 71. 77	6	58	1	7
15—25...	19. 26	2	29. 37. 44. 52. 56	5	7
25—35...	48. 63. 64. 65	4	37. 43. 47. 49. 52. 53. 56. 58. 59	9	13
35—45...	65. 68	2			2
Total		14		16	30

Thus, the disease lasted from twenty-five to thirty-five years in thirteen out of thirty cases; nine of them were those of females, who acquired it at or before the twentieth year of age. It lasted from five to fifteen years in six males, in whom it first appeared at or after the fortieth year of age. The shortest duration was in one female, aged thirty-four, who had symptoms of a cardiac affection. The longest was in two males, aged sixty-five and sixty-eight; of whom the one had lived for a long time in India, the other was a surgeon, who practised in Devonshire.

In most of the cases, death occurred very rapidly; but except in a few of them, the causes could not be ascertained. In five, it took place, as already mentioned, during the paroxysms. One patient was sent to Australia for the sake of his health, and died there aged nineteen. Another, aged twenty-six, had for the same purpose gone to the Cape, and died in Cape Town hospital, a few hours after admission, from some acute febrile disease. A third, aged sixty-five, died with symptoms of chronic myocarditis. Only two cases were fully observed.

Mr. R., aged sixty-five, came to town in a state of the greatest marasmus. On the 3rd of May, 1887, he was seized with a rigor, which was soon followed by signs of acute pneumonia of both upper lobes, and he died within ten days.

In the following case the diagnosis remained obscure.

Mrs. W., aged thirty-five, presented herself on January 16th, 1881, complaining of attacks of Asthma, which for several years had returned at least three times, days and nights. She could obtain relief only by inhaling the fumes of a French specific. She also had a troublesome cough, with frothy expectoration, occasionally tinged with blood, and a persistent running from her nose. The slightest exertion brought on dyspnœa, so that she would never venture any distance from home without carrying her pet remedy with her. Her appetite was good, her bowels generally acted several times daily, and her catamenia were regular.

She stated that she had an hereditary disposition to the disease, the origin of which she traced to whooping cough when five years old. Since then, she is said to have had, several times, such severe forms of acute bronchitis as caused the greatest anxiety with regard to her recovery.

Patient was rather tall and inclined to obesity. Her eyes were projecting, and her face was jaundiced and cyanotic. The nostrils were red and inflamed from the constant inhalation of the irritating smoke. The nose was pervious, and the pharynx and tonsils were the seat of a hyperplastic catarrh.

The thorax was small, the sternum arched, and the spine straight. Resp. 24. Expansion equal on both sides. Except a slight dulness in the right infra-clavicular region, there was hyper-resonance on percussion of both sides in front, extending to the sixth ribs. All over the right, a loud rasping noise was heard; on the left, there was complete silence, even after repeated coughs. On both sides, behind, the resonance was impaired, and there were sub-mucous rhonchi from apex to base.

The cardiac impulse could not be seen nor felt. The cardiac area could not be made out. The sounds were clear but feeble. Pulse 96, very small and soft.

The tongue was slightly furred, the abdomen greatly distended and tympanitic. The liver could not be felt nor percussed. The stools were said to be loose, but of normal colour. The legs were not œdematous.

The sputa contained the characteristic constituents, and the urine was normal.

Mrs. W. gradually recovered, so that she was able to leave off the smoking powder.

On June 13th, 1881, she had, what she called, an attack of "hay-asthma." After a few days of malaise, she had an intense burning sensation at the back of the throat. Her eyes were blood-shot and smarting; the face was swollen; the sneezing incessant; the pharynx and the velum palati were of white colour and œdematous. On the right side, in front and behind, there were abundant sibilant, on the left in front, sonorous rhonchi, and behind was complete silence.

In November of the same year, she had a very similar attack, which could in no way be connected with hay, nor with the hay season.

From that time, she was quite free from Asthma.

On July 4th, 1884, I was requested to see her, and was informed of the following: The week previously she had had a severe rigor, which was soon followed by intense pain in the back, extending to the legs, so that she imagined that she had somehow contracted an attack of small-pox. For a day or so, she had been unable to leave her bed, but afterwards, though feeling very ill, she had nevertheless attended the evening *fête* at the Botanical Gardens. The day before she was seen, she had gone to a dinner party; but her hostess had remarked how ill she looked, and how burning hot her hands were. She had, however, to leave the dinner table, and had spent a very restless night.

The patient was in the recumbent posture, and her face, instead of presenting its habitual jaundiced aspect, was of an ashy grey colour. She was rather apathetic, and complained only of intense headache. The skin was hot and dry, and there was no eruption of any kind. Temp. at 4.30 p.m. 104.5° F., R. 28, P. 120, very small and soft, but regular. The pupils were narrow, equal, and acted normally. The tongue was dry and slightly coated. In the left infra-clavicular region, there were absolute dulness, increased resistance, loud bronchial respiration, and bronchophony. The rest of the chest was hyper-resonant on percussion, and sonorous rhonchi were heard all over. There was neither subjective dyspnœa, nor cough, nor expectoration. The abdomen was very tympanitic, and neither the liver nor the spleen could be felt and percussed. Deep pressure in the right ileo-cæcal region was painful, and there was some gurgling. There was no œdema of the ankles. Motility and sensibility were intact. Her bowels had not acted for two days, but she had passed small quantities of a highly concentrated urine which contained no albumen. Cold applications to the head and the abdomen.

July 5th. A dose of three grains of Calomel had, during the night, produced six poultaceous stools of the colour of yolk of egg. About ten ounces of highly concentrated urine were passed altogether. The night had been restless. Temp. at 10 a.m. 103.6° F. P. 120, very small and soft. R. 28. The same state as yesterday. There were no signs of infiltration of the lungs, no roseola nor perceptible enlargement of the spleen.

At 9 p.m., temp. 104° F. The same typhoid state. The bowels had, again, acted several times, but there was no improvement. A fair amount of stimulants had been given during the day.

July 6th, 9 a.m. The night had been restless. Temp. 102.2° F. P. and R. as before. Increasing apathy, the same complaint of headache. Tongue very dry. Picking of bedclothes; otherwise, no change in the objective signs.

At 4 p.m., temp. 101.5° F. The quantity of stimulants was increased. Three doses of Musk with Ammon. Carb. āā five grains, at short intervals.

At 11 p.m. Collapse. Temp. 94° F. Face cyanotic. Sensorium became free. She sat up in bed and now complained of dyspnœa. The cardiac dulness was decidedly increased towards the right side, but the sounds were very feeble. On her particular request, she first inhaled the fumes of the powder which had been used in former years; and as that inhalation produced no relief, she had a small dose of morphia. Towards morning she slept two hours.

July 7th, 10.30 a.m. Temp. 104° F. P. 130, very small, soft and irregular. Great restlessness, increased weakness, and apathy. Stools and water were passed in bed. Stimulants, Musk and Ammon. Carb. Cold ablutions.

At 9 p.m. temp. 105.2° F. The same state. No change in objective signs.

July 8th, 8 a.m. Temp. 104·6° F. Coma. The temperature rose again in the evening to 105° F., and on July 9th, at 8 o'clock, to 106° F. At noon Exit. Lethal. P. M. was not permitted.

EFFECTS OF TREATMENT.

The question only now remains, whether or not Bronchial Asthma can be favourably modified by treatment.

According to the received opinion, it is the most intractable of all diseases, and the results generally obtained go far to justify that view.

But it is necessary to consider, before arriving at any conclusion, the circumstances in which, as a rule, the patients are seen, and the difficulties there are in the way of successfully managing a given case.

At the outset, there is the constitutional disposition at the root of the disease. There are probably some individuals, who are more than others prone to react with a fibrinous exudation to any irritation of their respiratory surface. Whether that peculiarity be due to the frailty of their epithelium, or to the state of the walls, or of the contents, of their vessels, is quite unknown. Possibly, the scanty number of mucous glands, which in the case above reported was especially remarked in the bronchial mucous membrane, may in some measure be responsible for it.

Thus, originally weighted, most asthmatics present themselves for advice only after years of suffering; after many futile attempts at treatment; and especially when for a long time their respiratory wants had been adjusted to their limited means of supplying them; so that the nutrition of every one of their organs had been considerably impaired in consequence.

But the chief difficulty lies in a different direction. The theory, long prevalent, that the "asthmatic tendency" is not of the least importance,[1] has gradually found its way to the public. The patients are convinced that all that is wanted is, to avoid the exciting cause of the Asthma, and failing that, something to mitigate the severity of the paroxysm. But, as according to the current doctrines, almost every thing or circumstance in life may at times be a provocative of the disease, an effective prevention is evidently impossible; the more so, in view of this disheartening element—that every precaution and the most careful calculation may unexpectedly be frustrated by the inherent "capriciousness of the disease." Nor is there any remedy that will afford a more than transient benefit, where the obstacle to respiration is entirely mechanical.

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[1] Hyde Salter, *Op. cit.*, p. 181.

Despondent, therefore, as to the results of treatment—for which indeed their experience is a sufficient warrant—asthmatics bear their troubles with an incredible amount of resignation. They are content to pant at the least exertion; to inhale the fumes of some nostrum every hour, day and night; and, so long as they obtain a temporary relief, they look upon their actual state as perfectly normal. They seek advice only when their usual remedies have lost their effects, and after they have tried in vain all those contained in the trade lists of speculative druggists. And even then, their avowed object is to find out something similar to which they have hitherto been accustomed.

Few, indeed, submit to a rational treatment; and those, who do so, look almost forward to the working of miracles. Yet, considering on the one hand, the pathological conditions, and on the other, the available means, it is evident that improvements can only be a matter of time and the results of measures adapted to the existing indications. Where scope, however, is given, it is by no means hopeless to realise all reasonable expectations.

CHAPTER IX.

TREATMENT OF BRONCHIAL ASTHMA.

A. Treatment of the Paroxysms: Narcotics; Counter-Irritation; Dietetic and Symptomatic Measures; Emetics; Inhalations; Cardiac Stimulants.—B. Treatment of the Disease: (*a*) Chronic Form: Rest; Climate; Diet; Inhalations; Expectorants; Stimulants; Electricity; Pneumatic Treatment; Treatment of Complications. (*b*) Acute Form—Preventive Treatment.

A. TREATMENT OF THE PAROXYSMS.

IN the intense and protracted paroxysms, in which alone advice is sought, the suffering is so great that relief must be procured as speedily as possible. With that object in view, the causal indication can rarely, if ever, be met. There are no means, by which the perviousness of the air passages can be restored in the requisitely short time. Neither the resistance of the obstacle can be reduced, nor the *vis a tergo* be increased, as rapidly as the urgency of the case demands. Under the most favourable conditions, hours must elapse before a fresh cast is detached from its matrix, or before the old hyaline substances can be successfully acted upon. Yet, without some alteration in its size or in its cohesion, the available forces, though roused to their utmost activity, are powerless to displace or to expel the obstructive exudation.

The treatment is consequently limited to the suppression of the painful perception which arises from the retention of the sputum. Yet, although essentially symptomatic, measures of that kind have, nevertheless, a much wider influence than the one, for which they are directly designed. As previously explained, the subjective and objective phenomena of an asthmatic seizure are due, partly to the narrowing of the air passages, partly to the irritation which the pathological products exert upon the sensory fibres in the bronchial mucous membrane. In all hollow organs—in the lungs as much as, for instance, in the rectum or the bladder—the presence of a foreign body at or near the orifices causes forcible but unsuitable contractions of the muscles, which regulate their capacities. Hence, if what has above been described as "the bronchial tenesmus" is effectively allayed, the organism is, thereby, enabled not only to compensate for the temporary deficiency in the supply of the air, but also to initiate such changes in the obstacle itself as conduce to a more complete recovery of the respiratory function.

Narcotics.

In no way can the "bronchial tenesmus" be so rapidly and so safely subdued as by the subcutaneous injection of Morphia. In from ten to fifteen minutes after the operation—rarely longer, unless the absorption is unusually delayed by the weakness of the circulation—the terrible struggle for breath ceases. The wheezing diminishes or altogether subsides; the firmly contracted muscles relax; the flushed or cyanotic face assumes a more natural colour; the pulse at the wrist becomes fuller; the patient, who till then sat motionless, bent forwards and panting, is able to draw a deeper breath; and now relieved of the threatening suffocation, he leans back on his pillows to repose from what may truly be called the hardest of labours.

A corresponding change takes place in the signs of auscultation. No doubt the obstruction persists, but respiration becomes quicker and more superficial, so that the adventitious sounds are less distinctly heard, or are not at all produced.

There is, so far as my experience goes, no contra-indication. In the young and the old, in the male and the female, in those with seemingly healthy and in those with evidently diseased organs, Morphia may safely be injected to meet the pressing needs of the moment. All that is necessary is to carefully adjust the dose to the requirements of each case. The opinions of those who restrict it to the "purely nervous Asthma,"[1] or who proscribe it altogether,[2] are hardly justified by facts. The sole inconvenience that is likely to arise from it is, that even in a small dose it induces vomiting, especially in patients who, during their attacks, suffer a great deal from nausea; and then it is, for obvious reasons, favourable rather than otherwise.

The one danger from it lies in its striking and never-failing effects. The rapid transition from a state of impending asphyxia to one of almost complete euphoria is apt to confirm certain preconceptions on the nature of the dyspnœa; and thus, to lead to principles of treatment, of which the results are most disastrous. No protest can be emphatic enough against the theory that "the morphia must be continued till the tendency to the spasm is broken."[1] Nor does anything deserve so strongly to be condemned, as the habit of intrusting the patient or his nurse with the syringe and the solution. The best and the safest of remedies becomes, in such conditions, the most baneful of all.

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[1] Huchard, *De la guérison rapide des accès d'Asthme par l'emploie des injections hypodermiques de morphine, etc.*: *L'Union Médicale*, 1878, No. 86 *seq.*

[2] Lazarus, *Zur Asthmatherapie*: *Berl. klin. Wochenschr.* 1887, No. 7.

In the pusillanimous, who refuse to submit to an injection, there is no other alternative but to have recourse to Opium or to Chloral. Of either of these drugs a full dose must be given, to obtain the speedy relief of the dyspnœa. But their exhibition has many disadvantages, and is even not altogether free from risks. Opium is comparatively slow in its action, and its effects last long after the need for them has passed away. Chloral is said to paralyse the intra-cardiac ganglia, and may thus produce a rapidly fatal collapse. It is, therefore, to be avoided in all cases, in which the obstruction of the bronchi is very considerable, and in which the muscles of the heart are diseased. The possibility of its abuse is, here also, to be guarded against.

The subcutaneous injection of Salicylate of Cocaine has been recommended by Mosler[2] and his pupil.[3] But there are as yet no reports confirmatory of its alleged beneficial results.

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[1] Huchard, *Loc. cit.*

[2] *Ueber subcut. Injectionen v. Cocain. Salicyl. bei Asthma: Deutsche Med. Wochenschr.*, 1886, No. 11.

[3] Schaefer, *Zur Behandl. v. Asthma bronchiale mit subc. Inject. von Cocain. Salicyl.:* Diss. Inaug., Greifswold, 1886.

Counter-Irritation.

A mustard leaf or a linseed meal poultice applied to the chest occasionally procures some ease; and asthmatics are, as a rule, grateful for any little assistance that may be rendered to them in the hour of their sufferance. The slight hyperæmia from those applications, limited as it is to a few square inches of the skin, has no material influence upon the circulation in the lungs, nor upon the interchange of gases. Their efficacy merely consists in that the irritation of the one, and the weight of the other, prevent, so far as they are able, the forcible contractions of the respiratory muscles.

Dietetic and Symptomatic Measures.

There are cases in which one injection of Morphia meets not only the symptomatic, but, to a great extent, also the causal indication. The rest which it procures enables the organism to prepare for the removal of the obstruction. After a short sleep, a few pellets are expectorated; and to all appearances the disease is at an end. The chest feels freer than it probably did for some time previously, and no abnormal sign can be detected in it by the most careful examination. Here, the dyspnœa plainly manifests itself in the light of a salutary effort.

When the exudation, however, continues, or when its accumulated products are considerable or of large size, the injection produces only a temporary abatement. Within a few hours, it will be necessary to repeat the operation, as soon as the difficulty in breathing again becomes excessive.

The first respite, thus gained, is hardly the most suitable moment for attempts to restore the perviousness of the air passages. While the obstruction lasts, absorption is necessarily slow, and the effects of internal medication are consequently delayed. Preferable it appears to allow the organism to take, as it were, the initiatory step; and to assist it in doing so, by the adoption of such measures as directly or indirectly facilitate respiration.

Above all, attention should be directed to a sufficient ventilation of the sick room. The "inordinate" desire of asthmatics for fresh air is not the mere fancy, as Trousseau suspects, but the outcome of a natural and intelligible craving. There need be no fear of "catching cold."

In the chronic cases, with a great deal of cyanosis, and with a weak action of the heart, stimulants must at once be freely given.

Unless the bowels have, shortly before, freely acted, an aperient is very useful. Pills, however, should be avoided; for, partly by inattention, partly owing to the forcible respiration, they are apt to be sucked into the bronchi.

During the persistence of the intense dyspnœa the patient has neither desire for food, nor breath enough to swallow solids. Liquids, however, may be given in small quantities and often. Frequent sips will help to allay the great thirst, which is due not only to the copious perspiration, but also to the breathing through the mouth.

Emetics.

All the other remedies commonly recommended are useless. In the severe paroxysms a trial with them prolongs but needlessly the suffering of the patients. Whatever repute some of them may possess, has been gained only by their occasional successes, and exclusively in the protracted forms of the disease in which, owing to the habitually shallow respirations, the power of expectoration is greatly impaired. They may be of service in the cases of slight dyspnœa when the plug is relatively loose, so that it can be readily expelled by an increase of the expiratory force.

Thus, the vomiting induced by Ipecacuanha, Tartar emetic, and smoking Tobacco *ad nauseam*, may, in suitable conditions, procure some relief. The powerful compression of the chest in the act of retching at times suffices to clear the air passages of their loose contents. But it fails of that effect, if the obstructing substance is considerable and very tenacious. That the deep collapse which arises from smoking Tobacco, by those unaccustomed to it, alleviates the dyspnœa, is quite conceivable; but there are, happily, less barbarous and more effectual measures.

A dangerous amount of prostration is produced by Lobelia,[1] which is apparently rescued again from well-merited oblivion. In the small "anti-spasmodic" doses, in which the drug is usually given, it has, according to the unanimous opinions of those who have experience of its properties, no appreciable results whatever.[2] Children may take, in twenty-four hours, from two to four drachms of the tincture before it in any way affects their dyspnœa.[1] To rapidly obtain the desired benefit, large quantities of it are required; but they cause a train of symptoms—as giddiness, faintness, sickness, cold sweat, and complete muscular relaxation[2]—symptoms more formidable than the Asthma itself. Although a greater part of it may be removed by vomiting, still enough is kept back to maintain that pitiable state for a long time afterwards.

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[1] Dujardin-Beaumetz, *De l'Asthme chez les enfants et de son traitement par la teinture de Lobelia Inflata: Bull. gen. de Therap.*, 1886, 15 Oct.—Nunes, *De la teinture de Lobelia influe dans la therapeutique de l'Asthme: Bullet. gen. de Therap.*, 1886, 28 Fev.—Moncorvo, *Sur le traitement de l'Asthme, par la teinture de Lobelia à haute dose: Bull. gen. de Ther.*, 1886, 15 Mars.

[2] Hyde Salter, *Op. cit.*, p. 239.

Inhalations.

The fumes of burning Nitre paper and the smoke of cigarettes or powders, of which Strammonium and Nitre are the chief ingredients, act merely by the forcible cough, which they excite when inhaled. The sputum which at the time blocks the air passages may thus be expectorated, unless it be either too large or too firmly impacted; and on the removal of the obstacle, there is consequently a certain freedom of respiration.

Those inhalations, however, are necessarily powerless, where the obstruction is considerable; while in favourable circumstances, the relief which they afford is only of a transient nature. Most asthmatics are oliged to resort to their particular mixtures, several times both day and night; and there are many, who do so almost every hour of their lives.

But the advantages of such measures are more than counterbalanced by the injury which they inflict. An asthmatic accustomed to relieve his dyspnœa by inhaling

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[1] Dujardin-Beaumetz, *Loc. cit.*

[2] Hyde Salter, *Op. cit.*, p. 240.

Nitre or Strammonium can at once be recognised by his intensely inflamed nostrils and pharynx; and there can be little doubt, that the products of combustion and the charred particles which the smoke mechanically lifts into the air, are highly irritating to the bronchial mucous membrane. Denuded as that surface is of its epithelium, the exudation is thereby maintained, and the disease indefinitely prolonged. Besides, most of the preparations in use are the inventions of speculative chemists, and contain a variety of narcotics, which paralyse the action of the heart.

On the effects of Pyridine, recommended by Germain Sée,[1] it is impossible to form an opinion. Its odour is so offensive that no patient could as yet be induced to try it.

A few whiffs of Chloroform, Ether, Iodide of Ethyl[2] and Bichloride of Methylene[3] are said to be effective in the slight attacks. In the severe paroxysms, they are quite useless. Apparently a tolerance is soon established to Chloroform and Ether, so that enormous quantities are supported without, however, the least benefit. Thus, Trousseau relates the case of a patient under his care, whose dyspnœa was not relieved by sixteen ounces of chloroform. The daily inhalation of such large doses, ultimately produced a state of acute mania, resembling delirium tremens.[4] A patient, who came under my own observation, had previously consumed, day after day, ten shillings' worth of Ether. For a long time, he constantly lived in an atmosphere of it, without any avail to his Asthma.

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[1] *Du traitement de l'Asthme nervo-pulmonaire et de l'Asthme cardiaque par la pyridine: Bull. gen. de Therap.*, 1885, 30 Juin; et *L'Union Medic.*, No. 83.

[2] Germain Sée, *Du Traitement de l'Asthme par l'iodure d'éthyle: Bull. gen. d. Therap.*, 1878, 15 Fev.—*Bulletin de l'Acad. de Méd.*, No. 5-10.

[3] Demme, quoted by Oertel, *Handb. d. Respir. Therapie*, 1882, p. 311.

[4] *Op. cit.*, t. ii., p. 478.

Cardiac Stimulants.

Such remedies as Nitrite of Amyl,[1] Nitro-glycerine, Caffeine, and Belladonna, appeal to the circulation rather than to the asthmatic condition. Their benefit can be insignificant only, so long as the bronchi remains obstructed. The hot and strong coffee, and the hot alcoholic drinks act probably by the hot water, which stimulates expectoration. Quebracho[2] and Grindelia Robusta[3] have no effect whatever.

B. TREATMENT OF THE DISEASE.

Although the dyspnœa, whenever it occurs, claims and receives the first share of attention, it is, after all, merely a symptom, and often even an accident. At times, for reasons above stated, when its origin is almost unavoidable, it is, indeed, a legitimate object of special treatment. It is not so, however, in the great majority of instances, in which it persistently returns. In them, there is evidence of its being preventible—not, however, so long as the disease itself is considered unimportant, because it recedes, so to say, as seemingly insignificant.

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[1] Comp. Talford Jones, *Practitioner*, Oct. 1871.—Kitchen, *Americ. Journal of Insanity*, Oct. 1873.—Pick, *Das Amyl-Nitrit.*, 1877, p. 60 *seq.*—T. R. Frazer, *Edinb. Med. Journal*, July 1887; *Lancet*, July 9th, 1887; *Americ. Journ. of Med. Science*, Oct. 1887.

[2] Penzoldt, *Berl. klin. Wochenschr.*, 1879, No. 19.

[3] Henry Gibbons, *Americ. Journ. of Med. Science*, April 1867. —Alabneda Gibbons, *Philad. Med. and Surg. Report*, 1874, Sept. 19th.

The aims of a rational treatment are, consequently, obvious. If there are no means of checking the pathological process, its products, at all events, must not be allowed to accumulate, and those which have already collected must be removed, as soon as practicable. For, otherwise, the exudation retained at its place of formation produces, in its subsequent onward course, the transient obstructions of the main bronchi; while its presence moving, as it were, in a vicious circle, not improbably contributes to maintain and to spread the peculiar morbid conditions.

(*a*) CHRONIC FORM.

The return of the dyspnœa at short intervals is, in itself, the surest proof that secretion has accumulated somewhere in the air-passages. Physical diagnosis confirms, as a rule, the supposed existence of a more or less extensive obstruction of the bronchi; and where the signs which it furnishes are marked, they are, indeed, of great value. But its less characteristic or even negative results allow no safe inference to the contrary; for the partially or completely occluded tubes often lie amidst comparatively healthy pulmonary tissues, or at some distance from the surface; and withal, the tidal current of air is so feeble that it cannot pass the obstacle nor produce sonorous vibrations.

Rest.

In cases of that kind, absolute rest is the first requisite. The suggestion will generally meet with a great deal of opposition on the part of the patients. The accommodation of their reduced respiratory wants to their limited means of supply has established a sort of spurious health —*i.e.*, the performance of the function without conscious effort; and they are, consequently, averse to give up, for a time, either their business or their pleasure. But it is evident, that an organism working under such conditions has all its energies taxed to the utmost to provide for the necessities of the moment; but that, without some surplus, there can be no repair. Nor is there any validity in the argument, that the dyspnœa occurs with its greatest frequency just during the rest at night. The circumstances are hardly parallel. In that case the detachment of the exudation has been affected by the exertion on the preceding day, as shown by what are commonly called the premonitory symptoms; and no doubt, the repose and the sleep contribute their own share towards the subsequent results. But, though it cannot be too often repeated that the dyspnœa is in the end a salutary effort, still it is an exceedingly painful one, and by all possible means to be avoided.

The benefit, chiefly to be derived from rest, is that the precipitate impaction of a plug is thereby prevented. For behind the obstacle, at its seat of formation, changes go on continually. The imprisoned air gradually disappears, and there is developed a more or less complete atelectasis, accompanied by serous exudation, which invariably takes place when the vessels remain for a long time dilated and the circulation in them is slackened. Thus constantly bathed, as it were, in an alkaline fluid, the fibrinous masses are dissolved. Their cohesion is diminished; perhaps a part of them is reabsorbed. To some extent, the tube is again opened up, admitting the access of air. But still the *vis a* *tergo* is slight, and the onward progress of the exudation correspondingly slow. The more time the rest, with its superficial respiration, allows for the passage of the sputum, the less its size and resistance become; while the deeper breathing on exertion, forcibly and rapidly, effects its removal.

The patients may in that way be spared a great deal of needless suffering. But there are other advantages also. Their confidence in any rational treatment would at once be shaken by the return of the dyspnœa; for they can hardly be expected to appreciate the niceties of pathology. To them, the attack is the disease which plainly seems uncontrollable. Moreover, it demands immediate relief, and whatever measures be adopted for that purpose, they cannot be said to be indifferent.

Climate.

Asthmatics are promised, in books, that some places will afford them complete immunity from their dyspnœa. But—possibly owing to the endless capriciousness "of the disease"—there is no indication to guide in the selection of the one the most likely to suit; and there is no other means of finding it, but by a diligent search.

The hope, thus roused, is the outcome of a settled theory, according to which the climate of any locality has a variable and mysterious effect upon Asthma: causing it in some, and relieving it in others. The idiosyncrasy of the individual is said to account for so strange a phenomenon. But what is chiefly remarkable is, that the foul air of crowded cities is believed to have a curative influence in most instances. Whether the facts justify any such inference, is a totally different matter. My own experience does not incline me to that view. Only one case of the kind came under my own notice. It was that of a gentleman in affluent circumstances, who, on the entreaty of a medical friend, had gone to reside in the immediate vicinity of the Seven Dials. After some time he returned home—a sadder but a wiser man.

No useful purpose can be served in discussing the current stories on the subject. Only possibilities could be suggested, unless all the circumstances of the cases were known. No one would venture to deny that, in the same patient, Asthma may not arise at one place, and subside at another. But there is no reason for assuming, that the air of the places is alone responsible for those events; and that there are no other factors more likely to be concerned in their origin and their subsidence. The whole theory is only a hasty conclusion, based on the flimsiest of grounds. Instances like the following are probably not infrequently met with in practice: I was once consulted about a lady, who had been urged to live on the third floor of a certain hotel. The sole reason, upon which that advice was given, was that an asthmatic American had spent there a comfortable night.

Nothing, perhaps, so forcibly shows the absurdity of that theory as the following case, reported by Trousseau[1]:—

A patient at St. Omer, who had for a long time suffered from Asthma without obtaining any relief, had made repeated appointments with Trousseau for a consultation, but was invariably prevented from keeping them by his inability to move. At last he made an effort to start; but he was so ill, that he had to be carried to the railway station.

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[1] *Op. cit.*, t. ii., p. 450.

There already, his breathing became somewhat easier. Shortly after his arrival in Paris, and after having undergone a few days' treatment, his Asthma subsided. Trousseau was too modest to attribute the success to his management of the case, but suspected, that the air of Paris had produced the favourable result. An excursion into the country, curiously enough, brought back an attack of Asthma, while the return to Paris caused it again to disappear.

After some time, and apparently cured, the patient went back to St. Omer. But shortly afterwards, his Asthma reappeared. He was then as bad as ever. Later on he came on business to London; and here again he had no dyspnœa. On returning home, he had another relapse.

The facts were duly communicated to Trousseau, who saw in them the confirmation of the theory on the specific influence of the air of large cities; and he therefore insisted that the patient should forthwith leave St. Omer, and settle either in Paris or London. On inquiry ten years later, however, Trousseau learnt, that his advice had been disregarded. The patient had remained at St. Omer; but for five years he had been quite free from Asthma.

In spite of all, that may be held to the contrary, there can be no doubt, that the kind of air which is best for general health is best also for asthmatics. They must, however, be in a position to benefit by it. It can avail them but little, if its access to the respiratory surface is impeded by the obstruction of the air passages. One might as well expect to cure a stricture of the œsophagus by placing before the patient a highly nutritious meal.

So long, therefore, as the bronchi are filled with secretion, the patient is better at home. A change of air would only tempt him to exertion, and thus bring back his dyspnœa. If his prejudices are very strong, he would, as soon as he was able to travel, leave the place, as one that did not suit him. But even if, for some reason or other, he be prevailed upon to stay, he would, as it generally happens, relieve his depuratory attacks by inhaling irritating smoke, so that the advantages to be derived from the superiorities of the climate are almost entirely lost.

Diet.

A mixed diet is obviously the best, provided there is no contra-indication on the part of the digestive organs. As a rule, the appetite and the digestion of asthmatics are good. The feeling of distension, which is frequently complained of, is due partly to their habit of breathing through the mouth and thus sucking air into the stomach ; partly to the slow absorption in consequence of their enfeebled circulation.

The ingestion of food invariably increases the respiratory wants, and it is therefore apt to produce dyspnœa where the breathing surface is considerably reduced by extensive obstruction of the bronchi. Hence, asthmatics generally suffer after meals.

The quantity of the food, rather than the quality, must be carefully regulated. Comparatively little is required in the state of rest in which the patients are proposed to be kept, and what is allowed them must be distributed into several small portions and given at regular intervals.

In these circumstances, the time at which the food may best be taken is unimportant. If breakfast is the meal which causes asthmatics the least inconvenience, it is because it comes after the nocturnal attack, or after the morning expectoration, when the air passages are comparatively free. As the day wears on, the secretion again accumulates within the larger bronchi, so that the difficulty of breathing is necessarily greater after lunch, and still more so after a late dinner.

Inhalations.

Besides the hygienic measures just referred to, a variety of medicines are more or less useful in effecting the removal of the accumulated morbid products. It would save, however, a great deal of disappointment to all concerned if, at the outset, both the aims of the treatment, and the nature of the available means were clearly understood. Above all, there is this to be remembered, that the tenacious hyaline masses resist to some extent even the strong reagents, that may be applied to them after expectoration. They are, in reality, foreign bodies, firmly, yet mechanically, attached to the respiratory surface, and in a great measure withdrawn from its physiological influence.

The hopes once entertained as to the benefits of inhalations have not been confirmed by experience. Although the pulverised particles penetrate to the ultimate ramifications of the lungs, still their quantity is too small to produce an appreciable effect. The solutions of chloride of sodium or of lime water, as they are commonly used, are certainly harmless, while the deliberate respirations during inhalation may, under suitable conditions, prove of advantage. Nitrate of silver has, of late, been recommended, and is said to have cured Asthma.[1] It is preferable, however, to refrain from active remedies; for, owing to the obstruction of the numerous bronchi, they are brought mainly into contact with the healthy mucous membrane.

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[1] Virchow's *Jahresber.*, 1887, vol. ii., p. 380.

Expectorants.

The one member of that class most frequently prescribed is Iodide of Potassium, which, no doubt, entirely owes its repute in the treatment of Asthma to its expectorant properties. Indeed, Germain-Sée,[1] who warmly recommends it, distinctly states that it liquefies the bronchial secretions, and thus greatly facilitates expectoration, which, in its turn, renders the air tubes again pervious, and thereby assists respiration.

So far as its physiological action is known, its exhibition in Asthma appears quite rational. Readily excreted from the mucous membrane of the bronchi, it escapes from the vessels in a stream of fluid, and serves to soften the fibrinous masses contained within them. As the tubes, however, are obstructed, the circulation in them is slow; consequently large doses of the drug are required to obtain the desired effect. According to Sée, at least thirty grains should be given in twenty-four hours, and if necessary, the quantity may be increased up to one drachm.

Various modes of administration have been suggested. Milk is a good vehicle for it. Dujardin Beaumetz orders a solution of about twenty grains in the ounce, of which he gives a dessert-, or, if necessary, a table-spoonful, in beer with meals. It acts, however, more rapidly if taken on an empty stomach, and freely diluted in warm milk. The addition of a few grains of chlorate of potash apparently hastens its excretion.

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[1] *Maladies du Cœur*, 1879, p. 403.

As a rule, it is badly borne, and a few doses of ten grains produce in most cases acute Iodism. Sée, on the contrary, states that there is a greater tolerance of it in Asthma, than in any other form of disease.[1]

Opinions concerning its value in Asthma are, however, divided. As a constituent of Green's and of Aubrée's mixtures, it had for a time a great notoriety as the most effective anti-asthmatic; and Trousseau was thus induced to try it in a number of cases. In some of them, he obtained results which no other medication had given him; in others it not only failed, but apparently aggravated all the symptoms.[2] Hyde Salter was only lukewarm in its favour, and thought that its frequent exhibition indicated "rather the estimate of the prescriber about it, than the utility to those who take it."[3] Germain Sée looks upon it as a specific, and he was disappointed by it only in three out of nearly two hundred cases.[4] Leyden testifies to its efficacy, but Curschmann[5] palliates its want of success by assuming, that in his cases it had not been continued sufficiently long.

Such different estimates, are however, quite intelligible; for all depends upon the circumstances in which it is given, and upon the results which are expected from it. In the cases, in which the secretion is sufficiently loose, the Iodide of Potassium may, by still more softening it, facilitate expectoration and thus prevent a return of the dyspnœa. In others, in which the exudation has not yet undergone regressive metamorphosis, it may serve merely to detach it from its place of origin, and thus by obstructing the main passages, produce dyspnœa. In still others, where the obstruction exists, it would but slowly and imperfectly relieve the dyspnœa.

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[1] *Maladies du Cœur*, 1879, p. 403.

[2] *Loc. cit.*, p. 477.

[3] *Op. cit.*, p. 303.

[4] *Ibid.*

[5] *Verhandl. d. Congr. f. Inn. Med.*, 1885, p. 248.

Almost all the patients who came under observation had previously taken Iodide of Potassium, some declaring "by the bucketfuls"; and their experience of its efficacy was by no means encouraging. Some, indeed, stated, that they had felt better whilst taking it; others, that they had received no benefit from it. But here also it is to be considered, that, on the one hand, the dyspnœa makes spontaneous intermissions, and that asthmatics, on the other, do not usually continue a remedy, unless it has a striking effect.

In those cases, in which it had been given, it invariably produced the expected results. But it by no means showed any superiority in its action over that of other expectorants.

From what has been stated above, it is unnecessary to add that if, in combination with narcotics, it relieves a dyspnœal paroxysm, the benefit obtained thereby is not entirely attributable to it. Given alone, in such circumstances, it would take at least ten hours, and would require a dose of more than two drachms, before the intense suffering is somewhat allayed. In the acute form of the disease, it is quite inadmissible, being apt to aggravate the inflammatory process.

The salt of Sodium may be substituted for that of Potassium in cases, where this is not tolerated. But its effect has been found to be unreliable.

Amongst the other expectorants, Ipecacuanha and Tartar Emetic appear to be the most useful. A weak infusion of the former with a very small dose of the latter may, as a rule, be relied upon to help in evacuating the air passages.

Apomorphia administered internally is quite inert. Children may take as much as one grain in less than an hour and be not affected by it in any way.

Where the bronchi are full of loose secretion, an emetic quickly and effectively clears them. Sulphate of Zinc or of Copper is preferable to any other, as both are speedy in their action and produce very little depression.

A longer experience of Jaborandi and its alkaloid Pilocarpine has fully confirmed the favourable opinion concerning them as expectorants.[1] They are especially indicated when the exudation is firmly impacted, and where something is wanted to start it, as it were. Nothing equals, in that respect, the subcutaneous injection of Pilocarpine. Such a powerful remedy, however, requires the greatest caution in its use. It is unsuitable in cases, in which the action of the heart is very weak.

The unpleasant after-effects of the hot infusion of Jaborandi may be avoided by the addition of a little whisky or brandy. Pilocarpine, given internally in doses of one-tenth or one-eighth of a grain, frequently causes slight frontal headache.

Stimulants.

In chronic cases and in debilitated individuals stimulants are indispensable. Their liberal exhibition is often more serviceable in clearing the bronchi than expectorants are. Brandy or whisky may advantageously be given in hot and strong coffee or in hot water.

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[1] *British Med. Journal*, June 19th and 26th, 1880.

Great benefit is also derived, in such conditions, from the use of Camphor in small doses.

The plan of treatment sketched above must be persistently carried out till the perviousness of the air-passages has been restored. When the chosen measures fully answer their purposes, their effect may be followed almost step by step. In cases free from complications, in which the right lower lobe, for instance, is imperfectly resonant and the seat of complete silence, the first sign of improvement is the appearance of faint clicking sounds. Gradually, rhonchi become audible over the obstructed parts, and as they move upwards, they are replaced by soft inspiration and expiration of equal lengths. Concurrently, the percussion note assumes a distinctly tympanitic sound. So long as the rhonchi are heard at the base, even slight exertion may readily produce dyspnœa; but as a rule there need be no such apprehension, when once they have reached the apex. In time, these symptoms also subside, and the normal state is apparently established.

Similar changes are observable in cases of considerable vicarious emphysema. On the removal of the accumulated secretion, the air is again equally distributed, and the over-distension yields.

Too much reliance, however, must not be placed on the negative physical signs, nor must all precaution be too soon relaxed. For otherwise the dyspnœa may unexpectedly return amidst seemingly perfect health. Such untoward accident not unfrequently arises from an overloaded bronchus, which, lying at some distance from the surface, may empty itself somewhat tardily. It soon passes off, however, but generally shakes the confidence of the patient. Absolute rest should, therefore, be maintained till, in addition to the cessation of the physical signs, the expectoration has entirely ceased during a few days.

Electricity.

The constant current applied to the vagus during a quarter of, or half, an hour is said to promptly relieve the dyspnœa, and to cause the emphysema, which may be present at the time, to at once subside. Eight out of sixteen cases have been radically cured by it.[1]

Bresgen[2] could not confirm its striking effect upon the paroxysm itself; but he, too, saw a marked improvement from it as regards the frequency of the disease.

Bernhardt,[3] on the contrary, is more cautious, suggesting that the question of its value could be decided only by a prolonged observation of the patient.

One lady, who came under my observation, had for a long time previously been treated with electricity, but had derived no benefit whatever from it.

Galvano-cautery of the nasal mucous membrane is also alleged to be a preventative of Asthma. It is supposed to be indicated, because the dyspnœa is ushered in by fits of sneezing. It recalls the former practice of applying caustic Ammonia to the pharynx—a practice which will hardly find imitators at the present day. The Ammonia produces some relief by the forcible cough which it excites; but it also causes œdema of the glottis, so that Trousseau and Rayer nearly lost their patients in consequence of the treatment. Electricity is certainly free from that danger.

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[1] Schaefer, *Asthma u. seine Behandlung: Deutsch. Med. Woch.*, Nos. 32 and 33: 1879.

[2] *Loc. cit.*

[3] Rosenthal u. Bernhardt, *Elektrizitätslehre, etc.*, 1884, p. 443.

Pneumatic Treatment.

The inhalation of compressed, the expiration into rarefied air, and a combination of both methods are also believed to be useful, both during the paroxysms and in the intervals between them.

Treatment of Complications.

In the cases, in which the disease has lasted for some time, there are generally numerous complications, partly accidental, partly of necessity connected with it; and though they are apparently not directly responsible for its return, yet they doubtless contribute towards it, by reducing the resistance of the body. So far as they are remediable, they require, therefore, special and approximate treatment.

Importance has of late been attached to the surgical treatment of the hyperplastic catarrh, and of the polypi, of the nose, whereby radical cures have been effected. It is quite conceivable that the secretion which in such conditions frequently accumulates in the naso-pharyngeal cavity may, for the reasons above stated, be the starting point of fresh attacks. In that way, the operation may have a beneficial effect; at all events, it restores the perviousness of the nose, and enables the breathing to be carried on *per vias naturales*. Still, one can but cordially subscribe to the opinion of Hack,[1] that the influence of the nasal mucous membrane upon Asthma has been grossly exaggerated.[1] In eighty-one of his cases, the Asthma, which had existed for years, subsided, while the hyperplasia of the nasal mucous membrane increased.

Polypi were observed in eleven, out of one hundred and eighty cases,—*i.e.*, in more than 6 per cent. In five of them, the operation for their removal was successfully performed by the most competent surgeons; the dyspnœa, nevertheless, returned with its former frequency and intensity. In the remaining six, there was, notwithstanding the persistence of the growths, a considerable improvement; so that the patients, who had, at one time, attacks almost daily, were free from them for several successive months. Two of them were persuaded to submit to operations, with the view of remedying the disagreeable feeling of stenosis, and if possible also, of guarding them against a recurrence of the disease. In the one, Mr. Arthur Durham removed several polypi, and completely destroyed, by galvano-cautery, the hyperplastic tissue of the nasal mucous membrane. Six weeks later, when the nose had been quite pervious, occurred the most formidable attack of Asthma. In the other, the disease had entirely ceased for seven months, but returned after the second operation, and terminated fatally in two days.

It is superfluous to add that the operations were not responsible, in those cases, for the return of the dyspnœa; but it is also certain that they cannot be credited with the improvement, if such happens to follow them.

The swellings of the joints, which Hack noticed in several of his cases after, were probably not caused by, the operation. They were observed also, where there was no kind of surgical interference.[1]

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[1] *Verhandl. d. Congresses f. In. Med.* 1885, p. 26.

(*b*) ACUTE FORM.

It is doubtful whether the process of exudation, if once established, can be successfully arrested. Large doses of Quinine have, perhaps, a more potent influence in that respect than any other remedy. The preparations of Mercury, which have been recommended in fibrinous bronchitis, have not had, when they were tried in Asthma, any marked effect; and large doses of Iodide of Potassium aggravate the disease in its acute form.

Fortunately, the tendency of the disease is to run a typical course. Its duration, its extent, and its intensity necessarily vary in different individuals. The quantity of the exciting cause, its actual virulence, and the temporary local and general dispositions of the patients are so many factors, unknown and inappreciable, which determine the result. Still, there is abundant evidence that the peculiar provocative soon exhausts its energy, unless an unsuitable interference supplies it with fresh pabulum or restores its activity. Irritating inhalations of the fumes of burning nitre paper or of strammonium facilitate its invasion of fresh portions of the surface, by destroying the resistance of the epithelium; for those products of combustion act chiefly on the healthy mucous membrane, their access to the seat of the disease being prevented by the obstruction of the affected bronchi. And the prolonged retention of the morbid products probably affords the best nutrient medicine for its development and proliferation.

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[1] *Vide supra*, p. 144.

Beyond guarding the patient against all noxious influences, the treatment can be only symptomatic. The extreme dyspnœa requires prompt relief by the hypodermic injection of morphia, to be followed as soon as practicable by some expectorant, for the purpose of moving the exudation. A weak infusion of Ipecacuanha, with a small dose of Tartar Emetic, appears to be the best of similar remedies, when the pulse is of good quality. In cases with feeble action of the heart, the Hydrochlorate or the Carbonate of Ammonia, or both combined, are preferable. Traube[1] recommends, in such cases, Ammon. Hydrochl., with an Infusion of Valerian. When the cyanosis is very considerable, and the pulse miserable, large doses of Camphor are the most useful.

Neither the relief from the dyspnœa, nor the fall of the febrile temperature, is a sure sign that the disease is at an end. So long as there are symptoms of stenosis, so long as the expectoration lasts, or so long as the face exhibits the least trace of cyanosis, the process either persists or the products are still retained. Notwithstanding all his pleadings, the patient must rest, and the above treatment be continued.

Preventive Treatment.

The exciting cause is so subtle, and probably so ubiquitous, that it is difficult to escape its influence. The main object of all preventive measures must, therefore, be to reduce the abnormal receptivity of the body. Consequently the constitutional anomalies which form the asthmatic tendency must, as far as possible, be counteracted. The obesity in some, and the anæmia in all cases, must be treated in the well-known manner. In marastic patients the exhibition of Iron is less efficacious than that of Arsenic, which possibly for this reason holds its place amongst the remedies for Asthma.

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[1] *Gesammelte Beiträge*, Band iii., 1878, p. 618.

Not the less important is it to confine the inflammatory process within harmless limits. There may be instances, in which it at once invades the entire surface; if so, they are apparently rare. As a rule, it gradually spreads from above downwards, so that, if suitable measures be taken in time, there is reasonable prospect of preventing its extension to the bronchial surface.

Strangely enough, the patients, persuaded that the dyspnœa alone is the disease, generally delay treatment till the disease has fully declared itself, although they frequently know of its approach by what they are in the habit of calling their "premonitory symptoms." Perhaps, when an extensive obstruction of the bronchi causes, in debilitated individuals, a considerable carbonic acid intoxication, efforts may be made to overcome the irresistible somnolence by sitting up at night and by drinking strong coffee. With what effect is notorious. As a rule, in spite of their ominous significance, fits of sneezing receive but little attention on the part of habitual asthmatics; they are popularly believed to be a cold, and, as such, not worth notice. Of late, however, their connection with the subsequent dyspnœa has been fully appreciated; only the current theory, by which the fact is explained, has led to no beneficial results. Painting the nasal mucous membrane with cocaine may indeed suppress the one manifestation of the inflammation; its cause remains nevertheless active, and its spreading is in no way prevented.

The profuse running from the nose, when the tissues of the nasal and pharyngeal mucous membrane are atrophic, and the more marked signs of irritation—such as sneezing, burning sensation at the back of the soft palate—when they are normal or hyperplastic, are indicative that the inflammatory process has set in. To arrest its extension, the surface not only of the nose and pharynx, but also of the tongue, must as soon as possible be thoroughly cleaned. A concentrated solution of Borax, with the addition of a few drops of Liqu. Hydrarg. Perchl. (P. B.), and perhaps, of a little Aqu. Flor. Samb. or of Eau de Cologne to cover the taste, best answers the purpose. If the patients are unable to gargle, a powerful spray must be employed. The nose must be washed out; and in order to effectually remove whatever secretion may have accumulated, or whatever micro-organisms may have settled there, the operations must be repeated at short intervals. A nasal douche may be used where it is at hand; otherwise it seems even preferable to pour the solution into a saucer or into the cover of a soap-dish and then to sniff it with head bent down and open mouth. The tongue also should be wiped with a soft silk pocket-handkerchief, dipped in the solution of Borax.

The local treatment is greatly assisted by the exhibition of Quinine, which, however, must be given in large doses. In the adult, not less than fifteen grains are required, and they are preferably taken at once, instead of being distributed over the day.

The same principles of treatment are applicable also to the abortive form of the disease, commonly known as "hay fever" or "hay asthma." The tissues being less accustomed to the influence of the morbific agent, the symptoms are more intense, and the antiseptic solution of Borax must consequently be sniffed, at first, every two hours, either tepid or nearly cool, and if possible used also for gargling. Later on, when the acute symptoms have somewhat abated, those operations need not be repeated oftener than three times a day. The irritation of the pharynx is best allayed by constantly sucking small lumps of ice, and that of the eyes by cold compresses, or by a spray of a concentrated solution of Borax.

But it is desirable to extend the preventive measures even further back. The ascertained facts that Asthma gradually develops itself, in the overwhelming majority of instances, in children of a rickety constitution, and, again, in those with a hereditary disposition, afford, at all events, some base for action. There is no need, in such circumstances, for looking upon every puny child as a possible candidate for the disease, and to anxiously subject him to a system of measures specially designed for the purpose. Nor is there any need for insisting here on the use of Iron and of Cod-liver oil, which, as a rule, are freely and even lavishly given. All that is necessary is, to pursue a more rational treatment, than is commonly adopted, in inflammatory affections of the air passages.

EXPLANATION OF PLATE I.

Fig. 1.—The frothy sputum during the attack allowed to settle on a sheet of black glass. It contains threads varying in length and thickness, all of them bent, but some distinctly spiral. Drawn with the aid of a pocket lens.

Fig. 2.—Two long spiral threads. $\frac{2}{1}$.

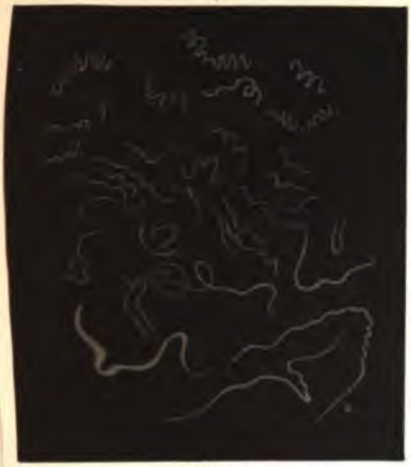
Fig. 3.—Portion of a solid cylindre, 8 cm. long.

Fig. 4.—Solid cast of a bronchiole. One branch of it straight, the other (*a*) spiral, consisting of several finer spirals. Natural size.

Fig. 5.—Solid piece ot sputum, natural size, of yellowish-white colour, containing on the surface delicate white lines.

The pieces represented in Figs. 2, 3, 4, and 5, were obtained from small, shapeless particles of sputum. They were teased by means of two fine camel-hair pencils on a glass slide in a solution of NaCl.

Fig. 6.—The piece represented in Fig. 5, examined with Powel & Leland's 1⅛" and drawn with the camera. It is composed of small, round, granular cells, and thick, hyaline fibres; on the surface of it, there are six threads (*a*—*f*), all of them cf different lengths and shapes.



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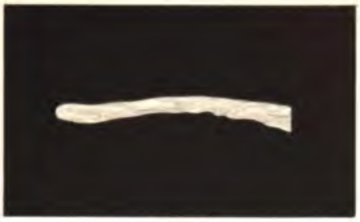
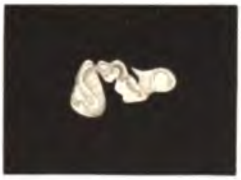


Fig. 3.

Fig 5.



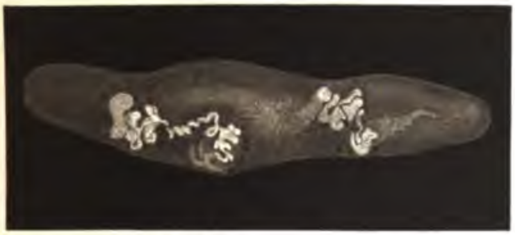
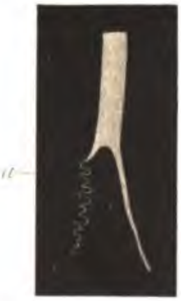


Fig 2.

Fig. 4.



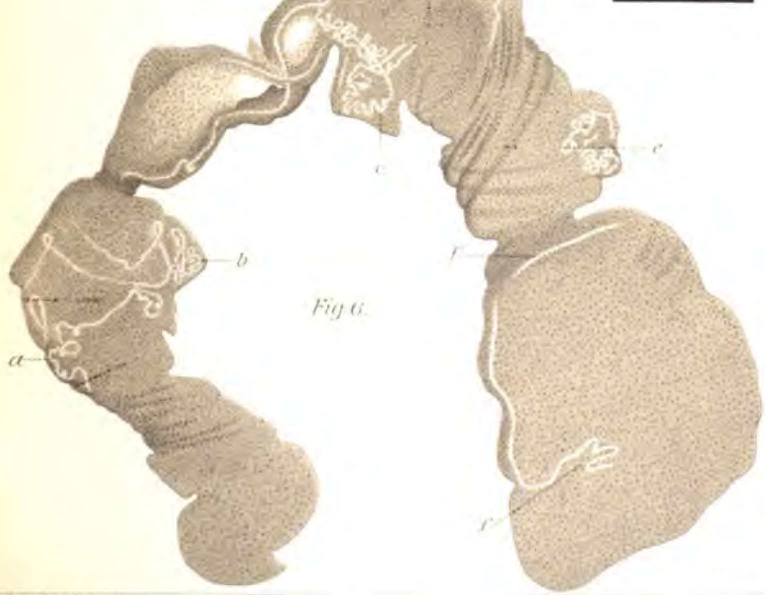


Fig 6.

Berkart Bronchial Asthma.

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EXPLANATION OF PLATE II.

Fig. 7.—The thread *a*, Fig. 6, examined with Zeiss A, Oc. 2, is here seen to be composed of several strands (*b*) closely twisted together. A glistening white line (*a a*) is contained in the centre above disappears, however, towards the middle portion, but reappears lower down. At *c c c* are heaps of small round cells with granular contents. At the end, *d*, the cord is frilled out into straight fibres.

Fig. 8.—The thread *c*, Fig. 6, examined with Zeiss A, Oc. 2. The twisting of the strands is looser than in Fig. 7, and the white central line, *a*, consequently more distinct.

Fig. 9.—Cast of a bronchiole, one branch apparently dilated and composed chiefly of cylindrical epithelium. Zeiss A, Oc. 2.

Fig. 10.—One of the thread-like formations as shown in Fig. 1. With Zeiss A, Oc. 2, it is seen to be a piece of pseudo-membrane twisted together.

Fig. 11.—A portion of the latter spread on a slide, dried and stained with methyline blue, according to Gram. It represents casts of infundibula and of alveoli, with concentric semicircles, *a a*, which have retained the dye.

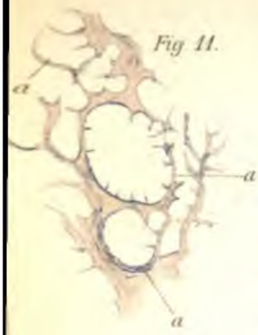
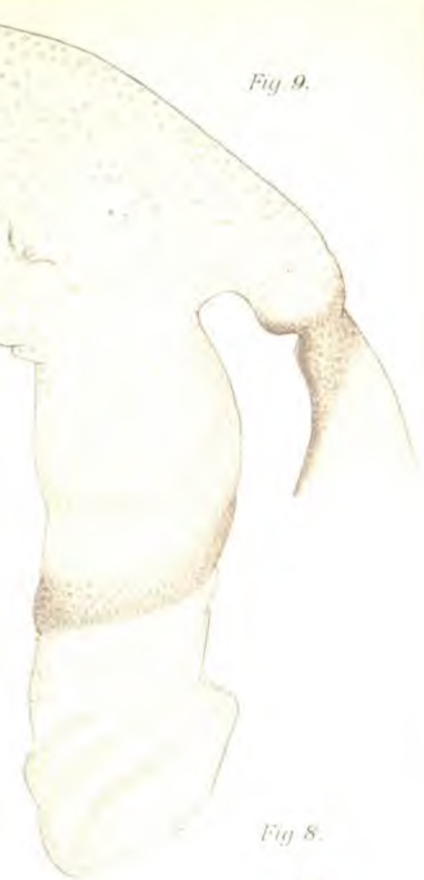


Fig. 11.



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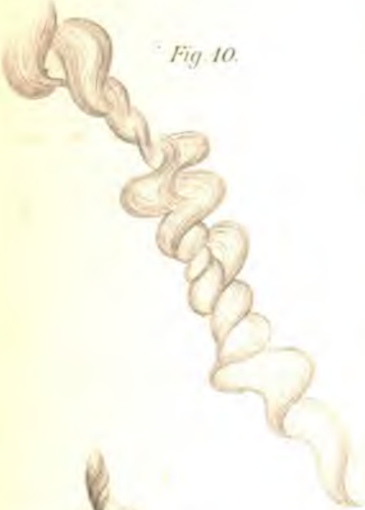
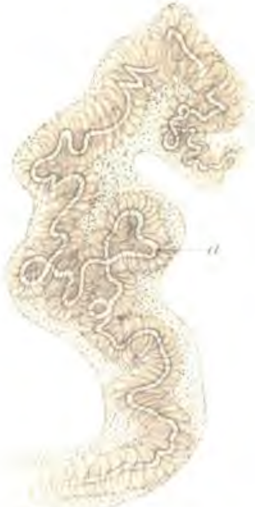


Fig. 10.



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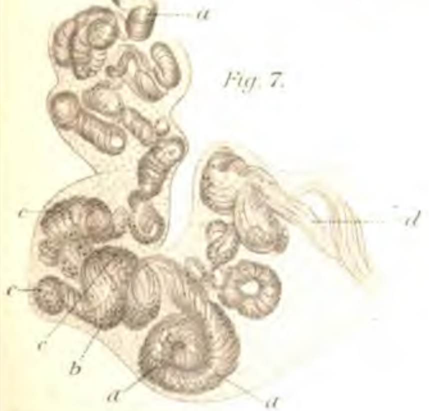


Fig. 7.

EXPLANATION OF PLATE III.

Fig. 12.—Capsule-cocci, some as diplo-cocci; others in chains of 2, 3, 4, and 5; also in tetrads. From the blue lines of Fig. 11. Zeiss Immers. $\frac{1}{12}$, Oc. 4. Tube 155 mm.

Fig. 13.—Three closely spun cords A, B, C, with fine lines projecting from the surface. Zeiss D, Oc. 2.

Fig. 14.—A small thread, stained. Zeiss A, Oc. 2.

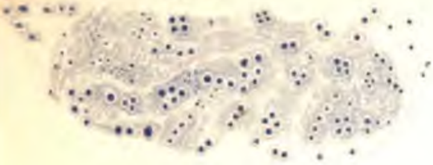
Fig. 15.—The same, examined with Zeiss Imm. $\frac{1}{12}$. Oc. 2. Tube 155 mm. It is seen to be composed of fine lines, and contains chains of streptococcus (*a*).

Fig. 16.—Deformed cylindrical epithelium. Zeiss D, Oc. 2.

Fig. 16A.—Cylindrical epithelium drawn out into threads and arranged in the figure of 8. Zeiss E, Oc. 2.

Fig. 16B.—Leucocytes in a state of disintegration containing micro-organism. From a cover-glass preparation of fresh sputum. Stained fuchsin and methyl blue. Zeiss Imm. $\frac{1}{12}$, Oc. 4.

Fig. 12.



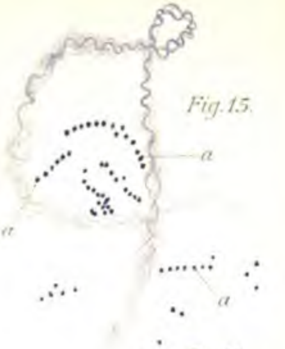
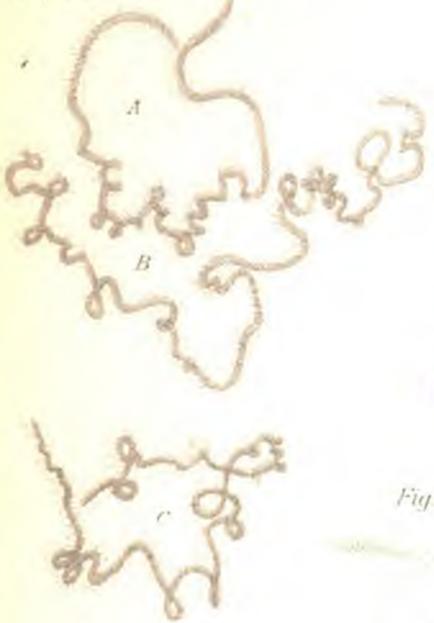


Fig. 15.

Fig. 13.



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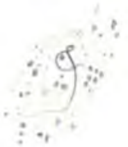

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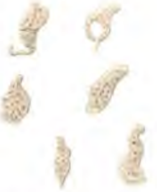


Fig. 16a

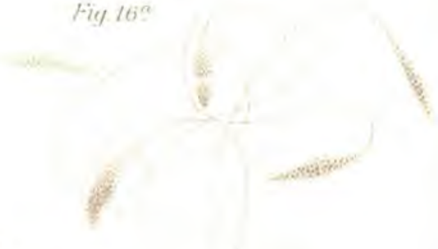
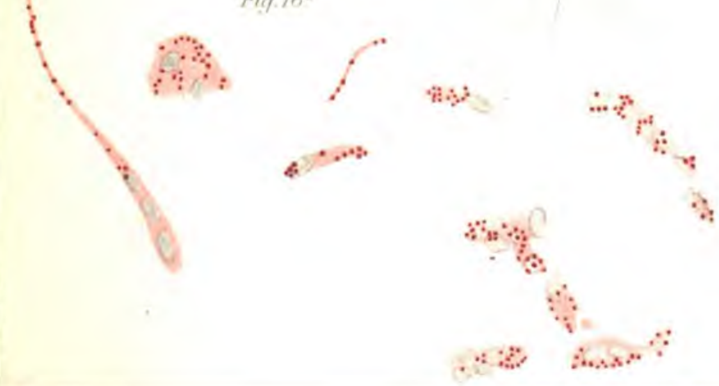


Fig. 16b



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EXPLANATION OF PLATE IV.

Fig. 17.--Cylindrical epithelium drawn out at one end, showing the various stages of the process. Zeiss D, Oc. 2.

Fig. 18.—Twisted thread with fibres, figure of 8. Zeiss D, Oc. 2. (From a photogram).

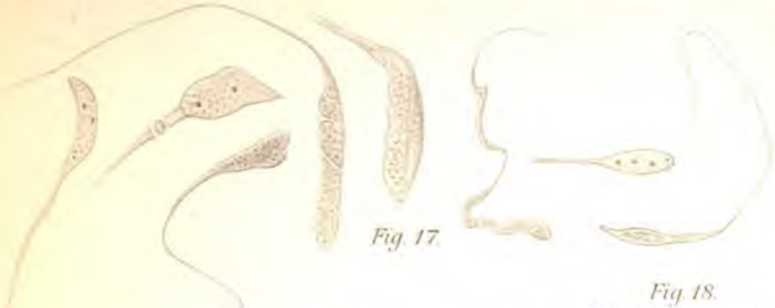
Fig. 19.—Isolated fibres of disintegrated epithelium; a roundish swelling at the end of one indicating the remains of the body of the cell. Small Charcot's crystals at the tops of two.

Fig. 20.—A long hyaline-looking thread in numerous coils, embedded in granular matter. Zeiss A, Oc. 2.

Fig. 21.—Two fine threads: A, fibres beginning to uncoil; B, twisted in the centre. Zeiss E, Oc. 2.

Fig. 22.—Cord, of which the strands are arranged in figure of 8, and uncoiled Upper portion breaking up into small rods and granules. Zeiss D, Oc. 2.

Fig. 23.—Charcot's crystals of various sizes.



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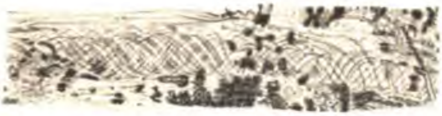
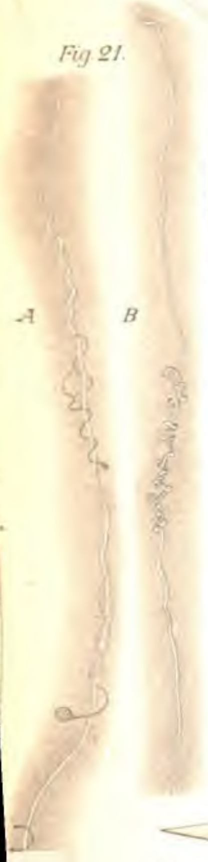
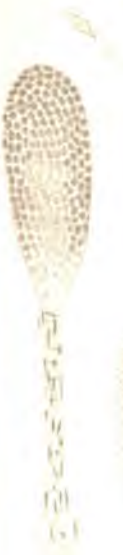


Fig. 18.

Fig. 21.





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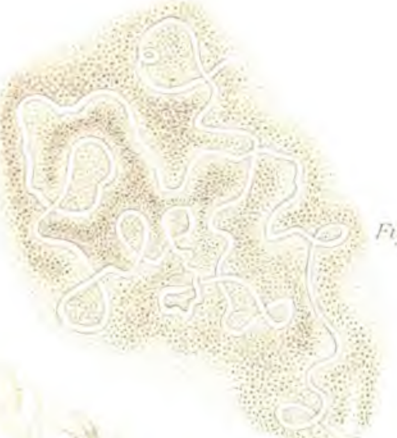


Fig. 20.

Fig. 22.

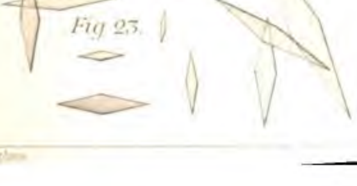


Fig. 23.

EXPLANATION OF PLATE V.

Fig. 24.—Hyaline fibres arranged in bundles. Zeiss D, Oc. 2.

Fig. 24A.—A bundle of delicate hyaline fibres matted together. Zeiss D, Oc. 2.

Fig. 24B.—A portion of the above examined with Zeiss Imm. $\frac{1}{12}$, Oc. 4. Tube drawn out. Hyaline fibres: *a*, thick; *b*, very delicate and undulating, resembling a spirochaete; *c*, breaking up into smaller and smallest rods; and between the meshes streptococci, *d*.

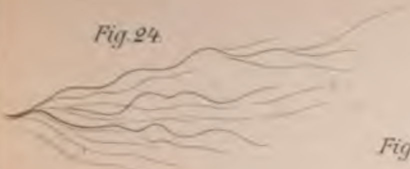
Fig. 24C.—Isolated threads varying in thickness, and the ends drawn out into irregular spirals. Zeiss Imm. $\frac{1}{12}$, Oc. 4. Tube 155.

Fig. 25.—A circular arrangement of fibres in Fig. 7, *c*. Zeiss D, Oc. 2.

Fig. 26.—Matrix of delicate fibres containing between their meshes chains of small bacilli, slightly rounded off at the poles with unstained centre. Gram. Zeiss Imm. $\frac{1}{12}$, Oc. 4. Tube 155.

Fig. 26A.—Combination preparation of a thread: *a*, matrix substance of longitudinal fibres varying in length and thickness; *b*, transverse fibres; *c*, disintegrated leucocytes with streptococci; *d*, chains of streptococci between the meshes of the fibres; *e*, bacilli in chains between the meshes; *f*, Charcot's crystals.

Fig. 24.



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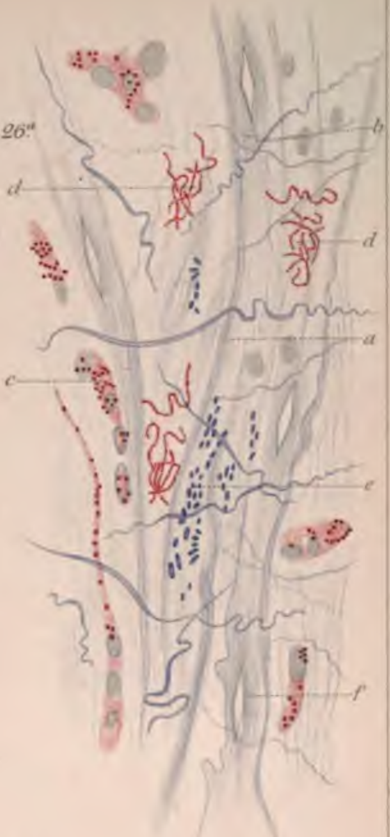
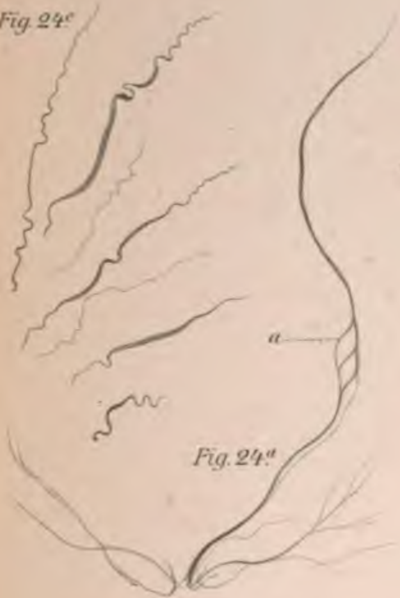


Fig. 24c.

Fig. 24a.



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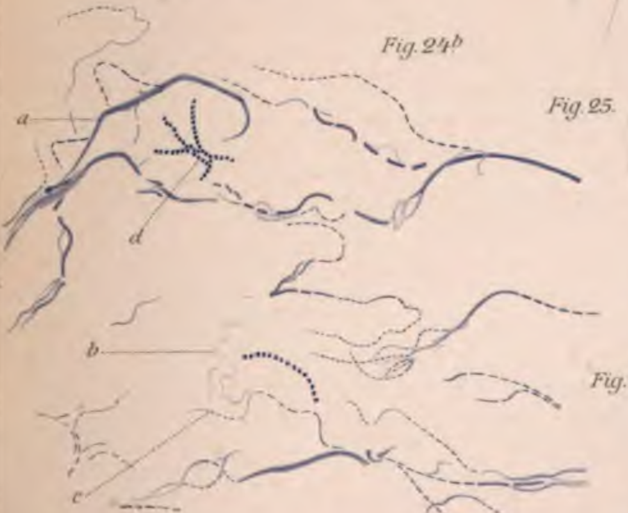

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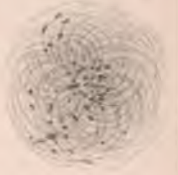

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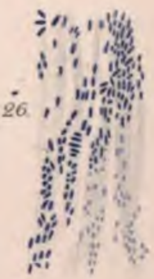

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EXPLANATION OF PLATE VI.

Fig. 27.—Chains of streptococci from cover-glass preparation. Gram. Zeiss Im. $\frac{1}{12}$, Oc. 2. Tube 155.

Fig. 28.—Small oval bacilli with deeply stained poles, from cover-glass preparation. Gram. Zeiss Im. $\frac{1}{12}$, Oc. 8. Tube drawn out.

Fig. 29.—Right main bronchus slit open and filled with spongy-looking substance. From the upper third, a small branch (*a*) is given off. The cylindre is chiefly made up of exfoliated cylindrical epithelium and hyaline fibres. Natural size.

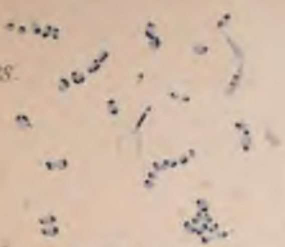
Fig. 30.—Transverse section through right main bronchus. The epithelium is exfoliated: *a*, deep ulcer extending closely to the cartilage *e*; *b*, fold of hyperplastic mucous-membrane projecting into the calibre and ulcerated at the free surface; *c*, mucous glands; *d*, artery. Powel & Leland, $1\frac{1}{2}''$, Oc. 2.

Fig. 31.—Transverse section through dilated bronchiole, with hyperplastic mucous membrane and containing masses of disintegrating exudation. Zeiss A, Oc. 2.

Fig. 32.—Section through lung substance showing emphysematous air vesicles and small bronchus obliquely cut, containing *a*, hyaline fibres stretched across.

Plate VI.

Fig. 28.



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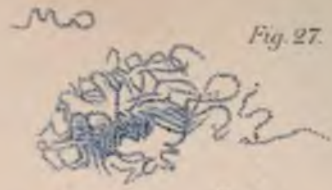

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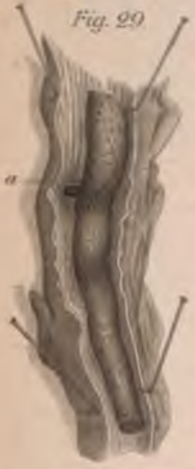

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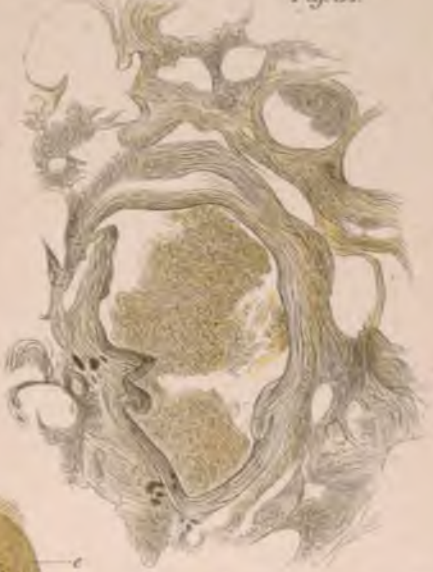

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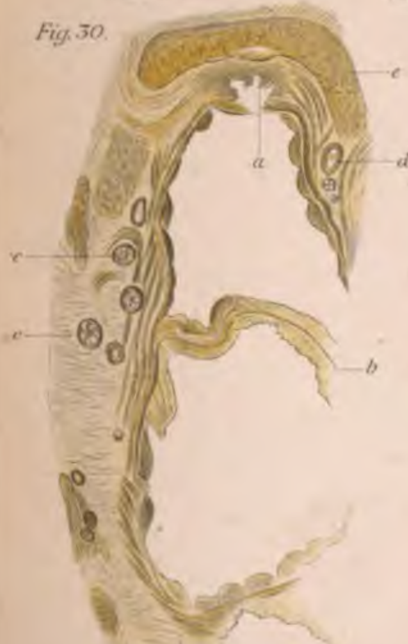

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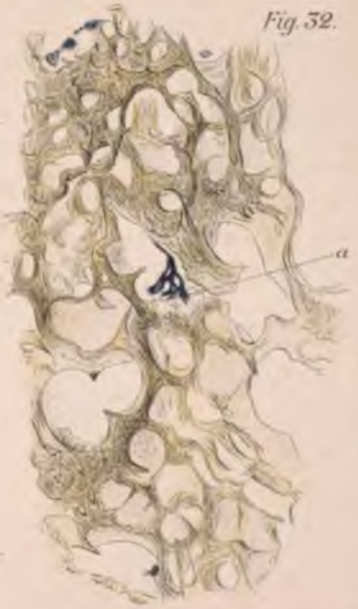

Berkart, Bronchial Asthma.

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EXPLANATION OF PLATE VII.

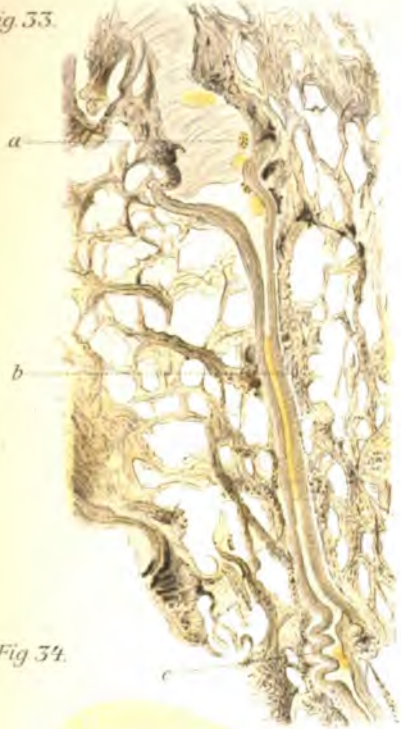
Fig. 33.—Longitudinal section through small bronchus and adjoining pulmonary tissues. The bronchus contains granular matter, and shows above irregular dilatation *a*; narrow portion *b*, in the centre; irregular spiral deformity of one wall, *c*. $\frac{20}{1}$.

Fig. 34.—Masses of streptococci contained in the exudation, *a*, in Fig. 33. Gram. Zeiss Im. $\frac{1}{12}$, Oc. 4. Tube drawn out.

Fig. 35.—Oblique section through small bronchus, showing dilatation, exfoliations of epithelium, and accumulation of detritus. Zeiss A, Oc. 2.

Plate VII.

Fig. 33.



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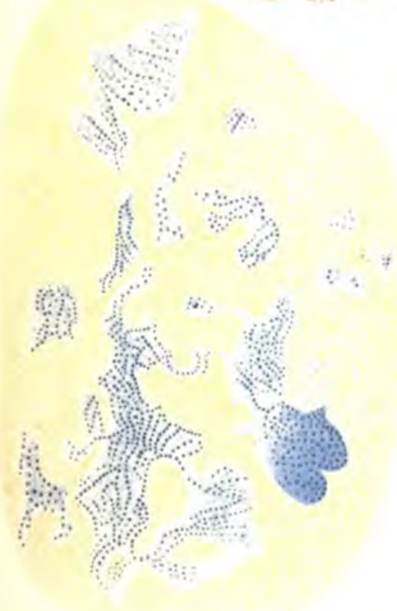
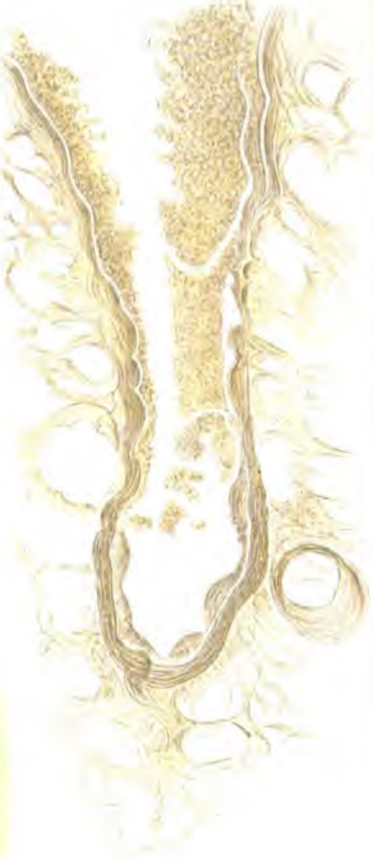


Fig. 35.



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